L3 ANSWER 9 OF 9 MEDLINE on STN
ACCESSION NUMBER: 2003491804 MEDLINE
DOCUMENT NUMBER: PubMed ID: 14568293
TITLE: Beta-glucan inhibits the genotoxicity of cyclophosphamide, adriamycin and cisplatin.
AUTHOR: Tohamy Amany A; El-Ghor Akmal A; El-Nahas Soheir M; Noshy Magda M
CORPORATE SOURCE: Zoology Department, Faculty of Science, Helwan University, Cairo, Egypt.
SOURCE: Mutation research, (2003 Nov 10) 541 (1-2) 45-53. Journal code: 0400763. ISSN: 0027-5107.
PUB. COUNTRY: Netherlands
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200312
ENTRY DATE: Entered STN: 20031022
Last Updated on STN: 20031219
Entered Medline: 20031203

AB The inhibitory effects of beta-**glucan** (betaG), one of the biological response modifiers, on the induction of chromosomal aberrations in the bone marrow and spermatogonial cells of mice treated with various anti-neoplastic drugs were investigated. beta-**Glucan** (100 mg/kg bw, i.p.) pre-treatment reduced the total number of cells with structural chromosomal aberrations scored after the treatment with **cyclophosphamide** (CP) (2.5 mg/kg bw, i.p.) adriamycin (ADR) (12 mg/kg bw, i.p.) and cis-diamminedichloroplatinum-II (cisplatin) (5 mg/kg bw, i.p.) by about 41.1, 26.9 and 57.7% in bone marrow and 44.4, 55 and 57.1% in spermatogonial cells, respectively. This protective effect of beta-**glucan** could be attributed to its scavenging ability to trap free-radicals produced during the biotransformation of these anti-neoplastic drugs. Beta-**glucan** also markedly restored the mitotic activity of bone marrow cells that had been suppressed by the anti-neoplastic drugs. These results indicate that in addition to the known immunopotentiating activity of beta-**glucan**, it plays a role in reducing genotoxicity induced by anti-neoplastic drugs during **cancer** chemotherapy.

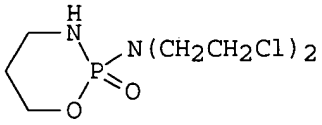
L3 ANSWER 9 OF 9 MEDLINE on STN
ACCESSION NUMBER: 2003491804 MEDLINE
DOCUMENT NUMBER: PubMed ID: 14568293
TITLE: Beta-glucan inhibits the genotoxicity of cyclophosphamide, adriamycin and cisplatin.
AUTHOR: Tohamy Amany A; El-Ghor Akmal A; El-Nahas Soheir M; Noshy Magda M
CORPORATE SOURCE: Zoology Department, Faculty of Science, Helwan University, Cairo, Egypt.
SOURCE: Mutation research, (2003 Nov 10) 541 (1-2) 45-53. Journal code: 0400763. ISSN: 0027-5107.
PUB. COUNTRY: Netherlands
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200312
ENTRY DATE: Entered STN: 20031022
Last Updated on STN: 20031219
Entered Medline: 20031203

AB The inhibitory effects of beta-**glucan** (betaG), one of the biological response modifiers, on the induction of chromosomal aberrations in the bone marrow and spermatogonial cells of mice treated with various anti-neoplastic drugs were investigated. beta-**Glucan** (100 mg/kg bw, i.p.) pre-treatment reduced the total number of cells with structural chromosomal aberrations scored after the treatment with **cyclophosphamide** (CP) (2.5 mg/kg bw, i.p.) adriamycin (ADR) (12 mg/kg bw, i.p.) and cis-diamminedichloroplatinum-II (cisplatin) (5 mg/kg bw, i.p.) by about 41.1, 26.9 and 57.7% in bone marrow and 44.4, 55 and 57.1% in spermatogonial cells, respectively. This protective effect of beta-**glucan** could be attributed to its scavenging ability to trap free-radicals produced during the biotransformation of these anti-neoplastic drugs. Beta-**glucan** also markedly restored the mitotic activity of bone marrow cells that had been suppressed by the anti-neoplastic drugs. These results indicate that in addition to the known immunopotentiating activity of beta-**glucan**, it plays a role in reducing genotoxicity induced by anti-neoplastic drugs during **cancer** chemotherapy.

L3 ANSWER 9 OF 9 MEDLINE on STN
ACCESSION NUMBER: 2003491804 MEDLINE
DOCUMENT NUMBER: PubMed ID: 14568293
TITLE: Beta-glucan inhibits the genotoxicity of cyclophosphamide, adriamycin and cisplatin.
AUTHOR: Tohamy Amany A; El-Ghor Akmal A; El-Nahas Soheir M; Noshy Magda M
CORPORATE SOURCE: Zoology Department, Faculty of Science, Helwan University, Cairo, Egypt.
SOURCE: Mutation research, (2003 Nov 10) 541 (1-2) 45-53. Journal code: 0400763. ISSN: 0027-5107.
PUB. COUNTRY: Netherlands
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200312
ENTRY DATE: Entered STN: 20031022
Last Updated on STN: 20031219
Entered Medline: 20031203

AB The inhibitory effects of beta-**glucan** (betaG), one of the biological response modifiers, on the induction of chromosomal aberrations in the bone marrow and spermatogonial cells of mice treated with various anti-neoplastic drugs were investigated. beta-**Glucan** (100 mg/kg bw, i.p.) pre-treatment reduced the total number of cells with structural chromosomal aberrations scored after the treatment with **cyclophosphamide** (CP) (2.5 mg/kg bw, i.p.) adriamycin (ADR) (12 mg/kg bw, i.p.) and cis-diamminedichloroplatinum-II (cisplatin) (5 mg/kg bw, i.p.) by about 41.1, 26.9 and 57.7% in bone marrow and 44.4, 55 and 57.1% in spermatogonial cells, respectively. This protective effect of beta-**glucan** could be attributed to its scavenging ability to trap free-radicals produced during the biotransformation of these anti-neoplastic drugs. Beta-**glucan** also markedly restored the mitotic activity of bone marrow cells that had been suppressed by the anti-neoplastic drugs. These results indicate that in addition to the known immunopotentiating activity of beta-**glucan**, it plays a role in reducing genotoxicity induced by anti-neoplastic drugs during **cancer** chemotherapy.

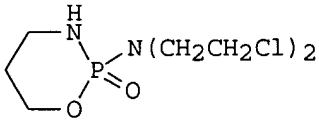
L3 ANSWER 7 OF 9 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1978:453298 CAPLUS
DOCUMENT NUMBER: 89:53298
TITLE: The synergistic effect of cyclophosphamide and glucan on experimental acute myelogenous and lymphocytic leukemia
AUTHOR(S): Di Luzio, N. R.; Cook, J. A.; Cohen, C.; Rodrigue, J.; Jones, E.
CORPORATE SOURCE: Dep. Physiol., Tulane Univ. Sch. Med., New Orleans, LA, USA
SOURCE: Proc. EURES Symp. Macrophage Cancer (1977), 188-201. Editor(s): James, Keith; McBride, Bill; Stuart, Angus. Univ. Edinburgh, Med. Sch.: Edinburgh, Scot. CODEN: 38BZA9
DOCUMENT TYPE: Conference
LANGUAGE: English
GI



I

AB In rats with Shay myelogenous leukemia, primary tumor growth was significantly reduced after administration of either **cyclophosphamide** (I) [50-18-0] (40 mg/kg, i.p., on days 3 and 6) or **glucan** [9012-72-0] (10 mg/kg, i.v. on days 3 and 6) alone compared to control rats. The most effective antineoplastic action, however, was evident with concurrent **glucan** and I therapy as denoted by a mean 97% decrease in tumor weight compared to control rats. In mice, increased survival after i.v. administered acute myelogenous leukemic cells was also observed in the **glucan** and I-treated group. I inhibited, to some degree, the **glucan**-induced hepatic granuloma. The degree of hepatic metastases was significantly reduced in both rats and mice by the conjoint use of I and **glucan**. Thus, **glucan** may be a valuable adjunct to conventional **cancer** chemotherapy.

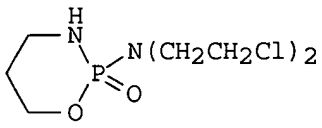
L3 ANSWER 7 OF 9 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1978:453298 CAPLUS
DOCUMENT NUMBER: 89:53298
TITLE: The synergistic effect of cyclophosphamide and glucan on experimental acute myelogenous and lymphocytic leukemia
AUTHOR(S): Di Luzio, N. R.; Cook, J. A.; Cohen, C.; Rodrigue, J.; Jones, E.
CORPORATE SOURCE: Dep. Physiol., Tulane Univ. Sch. Med., New Orleans, LA, USA
SOURCE: Proc. EURES Symp. Macrophage Cancer (1977), 188-201. Editor(s): James, Keith; McBride, Bill; Stuart, Angus. Univ. Edinburgh, Med. Sch.: Edinburgh, Scot.
CODEN: 38BZA9
DOCUMENT TYPE: Conference
LANGUAGE: English
GI



I

AB In rats with Shay myelogenous leukemia, primary tumor growth was significantly reduced after administration of either **cyclophosphamide** (I) [50-18-0] (40 mg/kg, i.p., on days 3 and 6) or **glucan** [9012-72-0] (10 mg/kg, i.v. on days 3 and 6) alone compared to control rats. The most effective antineoplastic action, however, was evident with concurrent **glucan** and I therapy as denoted by a mean 97% decrease in tumor weight compared to control rats. In mice, increased survival after i.v. administered acute myelogenous leukemic cells was also observed in the **glucan** and I-treated group. I inhibited, to some degree, the **glucan**-induced hepatic granuloma. The degree of hepatic metastases was significantly reduced in both rats and mice by the conjoint use of I and **glucan**. Thus, **glucan** may be a valuable adjunct to conventional **cancer** chemotherapy.

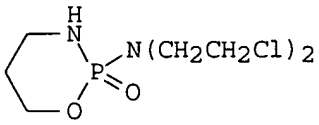
L3 ANSWER 6 OF 9 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1978:453416 CAPLUS
DOCUMENT NUMBER: 89:53416
TITLE: Enhancement of the inhibitory effect of cyclophosphamide on experimental acute myelogenous leukemia by glucan immunopotentiation and response of serum lysozyme
AUTHOR(S): Di Luzio, N. R.; Cook, J. A.; Cohen, C.; Rodrigue, J.; Kokoshis, P.; McNamee, R. B.
CORPORATE SOURCE: Dep. Physiol., Tulane Univ. Sch. Med., New Orleans, LA, USA
SOURCE: Progress in Cancer Research and Therapy (1978), 7(Immune Modulation Control Neoplasia Adjuvant Ther.), 171-82
CODEN: PCRTDK; ISSN: 0145-3726
DOCUMENT TYPE: Journal
LANGUAGE: English
GI



I

AB Tumor growth was reduced in rats receiving either **cyclophosphamide** (I) [50-18-0] or **glucan** [9012-72-0] alone compared to control rats. The most effective antineoplastic action, however, was evident with concurrent **glucan** and I therapy as denoted by a mean 97% decrease in tumor weight compared to control rats. Phagocytic activity of the reticuloendothelial system was subsequently evaluated after singular or combined administration of **glucan** and I. I abrogated the **glucan**-induced hyperphagocytic state even though interaction of these 2 agents was extremely effective in inducing tumor regression. Increased survival to i.v. administered acute myelogenous leukemic cells was also observed in the **glucan**- and I-treated group. I inhibited **glucan**-induced hepatic and pulmonary granuloma. **Glucan** elevated serum lysozyme [9001-63-2] concns. in both the presence and absence of I. **Glucan** may be a valuable adjunct to conventional **cancer** chemotherapy.

L3 ANSWER 6 OF 9 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1978:453416 CAPLUS
DOCUMENT NUMBER: 89:53416
TITLE: Enhancement of the inhibitory effect of cyclophosphamide on experimental acute myelogenous leukemia by glucan immunopotentiation and response of serum lysozyme
AUTHOR(S): Di Luzio, N. R.; Cook, J. A.; Cohen, C.; Rodrigue, J.; Kokoshis, P.; McNamee, R. B.
CORPORATE SOURCE: Dep. Physiol., Tulane Univ. Sch. Med., New Orleans, LA, USA
SOURCE: Progress in Cancer Research and Therapy (1978), 7(Immune Modulation Control Neoplasia Adjuvant Ther.), 171-82
CODEN: PCRTDK; ISSN: 0145-3726
DOCUMENT TYPE: Journal
LANGUAGE: English
GI



I

AB Tumor growth was reduced in rats receiving either **cyclophosphamide** (I) [50-18-0] or **glucan** [9012-72-0] alone compared to control rats. The most effective antineoplastic action, however, was evident with concurrent **glucan** and I therapy as denoted by a mean 97% decrease in tumor weight compared to control rats. Phagocytic activity of the reticuloendothelial system was subsequently evaluated after singular or combined administration of **glucan** and I. I abrogated the **glucan**-induced hyperphagocytic state even though interaction of these 2 agents was extremely effective in inducing tumor regression. Increased survival to i.v. administered acute myelogenous leukemic cells was also observed in the **glucan**- and I-treated group. I inhibited **glucan**-induced hepatic and pulmonary granuloma. **Glucan** elevated serum lysozyme [9001-63-2] concns. in both the presence and absence of I. **Glucan** may be a valuable adjunct to conventional **cancer** chemotherapy.

L3 ANSWER 5 OF 9 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1998:803767 CAPLUS
DOCUMENT NUMBER: 130:204804
TITLE: In vitro and in vivo hematopoietic activities of Betafectin PGG-glucan
AUTHOR(S): Patchen, Myra L.; Vaudrain, Tracy; Correira, Heidi; Martin, Tracey; Reese, Debrah
CORPORATE SOURCE: Alpha-Beta Technology, Worcester, MA, USA
SOURCE: Experimental Hematology (Charlottesville, Virginia) (1998), 26(13), 1247-1254
CODEN: EXHMA6; ISSN: 0301-472X
PUBLISHER: Carden Jennings Publishing
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Betafectin PGG-glucan is a novel β-(1,3)glucan that has broad-spectrum anti-infective activities without cytokine induction. Here the authors report that PGG-glucan also has both in vitro and in vivo hematopoietic activities. In vitro studies with bone marrow target cells from the C3H/HeN mouse revealed that although PGG-glucan alone had no direct effect on hematopoietic colony-forming cell (CFC) growth, when combined with granulocyte colony-stimulating factor (CSF) or granulocyte-macrophage CSF, it increased CFC nos. 1.5- to 2.0-fold over those obtained with CSFs alone. Bone marrow cells cultured for high-proliferative-potential CFCs in the presence of interleukin (IL)-1, IL-3, macrophage CSF, and stem cell factor (SCF), or cultured for erythroid burst-forming units in the presence of IL-3, SCF, and erythropoietin, also exhibited enhanced growth in the presence of PGG-glucan. The synergistic effect of PGG-glucan was specific and could be abrogated by anti-PGG-glucan antibody. The ability of PGG-glucan to modulate hematopoiesis in vivo was evaluated in myelosuppressed rodents and primates. C3H/HeN female mice were i.v. administered saline solution or PGG-glucan (0.5 mg/kg) 24 h before the i.p. administration of cyclophosphamide (200 mg/kg), and the recovery of bone marrow cellularity and granulocyte-macrophage progenitor cells was evaluated on days 4 and 8 after cyclophosphamide treatment. At both time points, enhanced hematopoietic recovery was observed in PGG-glucan-treated mice compared with saline-treated control mice. In a final series of in vivo expts., the authors evaluated the ability of therapeutically administered PGG-glucan to enhance hematopoietic recovery in cyclophosphamide-treated cynomolgus monkeys. Monkeys received i.v. infusions of cyclophosphamide (55 mg/kg) on days 1 and 2, followed on days 3 and 10 by i.v. infusion of PGG-glucan (0.5, 1.0, or 2.0 mg/kg). Compared with those in saline-treated monkeys, accelerated white blood cell recovery and a reduction in the median duration of neutropenia were observed in PGG-glucan-treated monkeys. These studies illustrate that PGG-glucan has both in vitro and in vivo hematopoietic activities and that this agent may be useful in the prevention and/or treatment of chemotherapy-associated myelosuppression.
REFERENCE COUNT: 45 THERE ARE 45 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 4 OF 9 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2000:324986 CAPLUS
DOCUMENT NUMBER: 133:202741
TITLE: Induction of apoptosis in human prostatic cancer cells with β-glucan (Maitake mushroom polysaccharide)
AUTHOR(S): Fullerton, Sean A.; Samadi, Albert A.; Tortorelis, Dean G.; Choudhury, Muhammad S.; Mallouh, Camille; Tazaki, Hiroshi; Konno, Sensuke
CORPORATE SOURCE: Department of Urology, New York Medical College, Valhalla, NY, USA
SOURCE: Molecular Urology (2000), 4(1), 7-13
CODEN: MOURFE; ISSN: 1091-5362
PUBLISHER: Mary Ann Liebert, Inc.
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Human prostate cancer PC-3 cells were treated with various concns. of the highly purified β-glucan preparation Grifron-D (GD), and viability was determined after 24 h. Lipid peroxidn. (LPO) assay and in situ hybridization (ISH) were performed to evaluate the antitumor mechanism of GD. A concentration-response study showed that almost complete (>95%) cell death was attained in 24 h with GD ≥480 μg/mL. Combinations of GD in a concentration as low as 30-60 μg/mL with 200 μM vitamin C were as effective as GD alone at 480 μg/mL, inducing >90% cytotoxic cell death. Simultaneous use with various anticancer drugs showed little potentiation of their efficacy, except for the carmustine/GD combination (.apprx.90% reduction in cell viability). The 2-fold elevated LPO level and pos. ISH staining of GD-treated cells indicated oxidative membrane damage resulting in apoptotic cell death. Thus, a bioactive β-glucan from the Maitake mushroom has a cytotoxic effect, presumably through oxidative stress, on prostatic cancer cells in vitro, leading to apoptosis. Potentiation of GD action by vitamin C and the chemosensitizing effect of GD on carmustine may also have clin. implications. This unique mushroom polysaccharide may have potential as an alternative therapeutic modality for prostate cancer.
REFERENCE COUNT: 26 THERE ARE 26 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 3 OF 9 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2003:808828 CAPLUS
DOCUMENT NUMBER: 140:138897
TITLE: β-Glucan inhibits the genotoxicity of cyclophosphamide, adriamycin and cisplatin
AUTHOR(S): Tohamy, Amany A.; El-Ghor, Akmal A.; El-Nahas, Soheir M.; Noshy, Magda M.
CORPORATE SOURCE: Faculty of Science, Zoology Department, Helwan University, Cairo, Egypt
SOURCE: Mutation Research (2003), 541(1-2), 45-53
CODEN: MUREAV; ISSN: 0027-5107
PUBLISHER: Elsevier Science B.V.
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The inhibitory effects of β- **glucan** (βG), one of the biol. response modifiers, on the induction of chromosomal aberrations in the bone marrow and spermatogonial cells of mice treated with various anti-neoplastic drugs were investigated. β- **Glucan** (100 mg/kg bw, i.p.) pre-treatment reduced the total number of cells with structural chromosomal aberrations scored after the treatment with **cyclophosphamide** (CP) (2.5 mg/kg bw, i.p.) adriamycin (ADR) (12 mg/kg bw, i.p.) and cis-diamminedichloroplatinum-II (cisplatin) (5 mg/kg bw, i.p.) by about 41.1, 26.9 and 57.7% in bone marrow and 44.4, 55 and 57.1% in spermatogonial cells, resp. This protective effect of β- **glucan** could be attributed to its scavenging ability to trap free-radicals produced during the biotransformation of these anti-neoplastic drugs. β- **Glucan** also markedly restored the mitotic activity of bone marrow cells that had been suppressed by the anti-neoplastic drugs. These results indicate that in addition to the known immunopotentiating activity of β- **glucan**, it plays a role in reducing genotoxicity induced by anti-neoplastic drugs during **cancer** chemotherapy.
REFERENCE COUNT: 48 THERE ARE 48 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 3 OF 9 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2003:808828 CAPLUS
DOCUMENT NUMBER: 140:138897
TITLE: β-Glucan inhibits the genotoxicity of cyclophosphamide, adriamycin and cisplatin
AUTHOR(S): Tohamy, Amany A.; El-Ghor, Akmal A.; El-Nahas, Soheir M.; Noshy, Magda M.
CORPORATE SOURCE: Faculty of Science, Zoology Department, Helwan University, Cairo, Egypt
SOURCE: Mutation Research (2003), 541(1-2), 45-53
CODEN: MUREAV; ISSN: 0027-5107
PUBLISHER: Elsevier Science B.V.
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The inhibitory effects of β- **glucan** (βG), one of the biol. response modifiers, on the induction of chromosomal aberrations in the bone marrow and spermatogonial cells of mice treated with various anti-neoplastic drugs were investigated. β- **Glucan** (100 mg/kg bw, i.p.) pre-treatment reduced the total number of cells with structural chromosomal aberrations scored after the treatment with **cyclophosphamide** (CP) (2.5 mg/kg bw, i.p.) adriamycin (ADR) (12 mg/kg bw, i.p.) and cis-diamminedichloroplatinum-II (cisplatin) (5 mg/kg bw, i.p.) by about 41.1, 26.9 and 57.7% in bone marrow and 44.4, 55 and 57.1% in spermatogonial cells, resp. This protective effect of β- **glucan** could be attributed to its scavenging ability to trap free-radicals produced during the biotransformation of these anti-neoplastic drugs. β- **Glucan** also markedly restored the mitotic activity of bone marrow cells that had been suppressed by the anti-neoplastic drugs. These results indicate that in addition to the known immunopotentiating activity of β- **glucan**, it plays a role in reducing genotoxicity induced by anti-neoplastic drugs during **cancer** chemotherapy.

REFERENCE COUNT: 48 THERE ARE 48 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 2 OF 9 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2005:259651 CAPLUS
DOCUMENT NUMBER: 142:291363
TITLE: Chemotherapeutic antineoplastic treatment
INVENTOR(S): Yvin, Jean-Claude; Vetvicka, Vaclav
PATENT ASSIGNEE(S): Fr.
SOURCE: U.S. Pat. Appl. Publ., 10 pp.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2005065111	A1	20050324	US 2003-668661	20030923
WO 2005027938	A1	20050331	WO 2004-EP10993	20040916

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.: US 2003-668661 A 20030923

AB Chemotherapeutic method for the treatment of cancer comprising administration of an effective amount of an antineoplastic agent in conjunction with an effective amount of a β-1,3 glucan is disclosed.

L3 ANSWER 2 OF 9 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2005:259651 CAPLUS
DOCUMENT NUMBER: 142:291363
TITLE: Chemotherapeutic antineoplastic treatment
INVENTOR(S): Yvin, Jean-Claude; Vetvicka, Vaclav
PATENT ASSIGNEE(S): Fr.
SOURCE: U.S. Pat. Appl. Publ., 10 pp.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2005065111	A1	20050324	US 2003-668661	20030923
WO 2005027938	A1	20050331	WO 2004-EP10993	20040916

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.: US 2003-668661 A 20030923

AB Chemotherapeutic method for the treatment of cancer comprising administration of an effective amount of an antineoplastic agent in conjunction with an effective amount of a β-1,3 glucan is disclosed.

L3 ANSWER 1 OF 9 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2006:66525 CAPLUS
TITLE: Soy isoflavone aglycone modulates a hematopoietic response in combination with soluble β-glucan: SCG
AUTHOR(S): Harada, Toshie; Masuda, Susumu; Arii, Masayuki; Adachi, Yoshiyuki; Nakajima, Mitsuhiro; Yadomae, Toshiro; Ohno, Naohito
CORPORATE SOURCE: Laboratory for Immunopharmacology of Microbial Products, School of Pharmacy, Tokyo University of Pharmacy and Life Science, 1432-1 Horinouchi, Hachioji, Tokyo, 192-0392, Japan
SOURCE: Biological & Pharmaceutical Bulletin (2005), 28(12), 2342-2345
CODEN: BPBLEO; ISSN: 0918-6158
PUBLISHER: Pharmaceutical Society of Japan
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Soy isoflavone aglycons (IFAs) have a wide range of biol. actions that suggest they may be of use in **cancer** prevention. A branched β- **glucan** from Sparassis crispa (SCG) is a major 6-branched 1,3-β-D- **glucan** in an edible/medicinal mushroom, Sparassis crispa, showing antitumor activity. We have previously reported that both oral and i.p. administration of SCG enhanced the hematopoietic response in **cyclophosphamide** (CY)-induced leukopenic mice. In this study, we investigated the hematopoietic response due to IFA in combination with SCG in CY-induced leukopenic mice. The oral administration of IFA in combination with SCG synergistically enhanced the number of white blood cells, and increased spleen weight Analyzing the leukocyte population by flow cytometry, the combination of IFA and SCG increased the number of monocytes and granulocytes in the spleen. Taken together, the combination of IFA and SCG synergistically provides the hematopoietic responses that are enhanced over IFA or SCG alone.
REFERENCE COUNT: 20 THERE ARE 20 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 2 OF 9 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2005:259651 CAPLUS
DOCUMENT NUMBER: 142:291363
TITLE: Chemotherapeutic antineoplastic treatment
INVENTOR(S): Yvin, Jean-Claude; Vetvicka, Vaclav
PATENT ASSIGNEE(S): Fr.
SOURCE: U.S. Pat. Appl. Publ., 10 pp.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2005065111	A1	20050324	US 2003-668661	20030923
WO 2005027938	A1	20050331	WO 2004-EP10993	20040916

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE,

SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG
PRIORITY APPLN. INFO.: US 2003-668661 A 20030923
AB Chemotherapeutic method for the treatment of cancer comprising administration of an effective amount of an antineoplastic agent in conjunction with an effective amount of a β-1,3 glucan is disclosed.

L3 ANSWER 3 OF 9 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2003:808828 CAPLUS
DOCUMENT NUMBER: 140:138897
TITLE: β-Glucan inhibits the genotoxicity of cyclophosphamide, adriamycin and cisplatin
AUTHOR(S): Tohamy, Amany A.; El-Ghor, Akmal A.; El-Nahas, Soheir M.; Noshy, Magda M.
CORPORATE SOURCE: Faculty of Science, Zoology Department, Helwan University, Cairo, Egypt
SOURCE: Mutation Research (2003), 541(1-2), 45-53
CODEN: MUREAV; ISSN: 0027-5107
PUBLISHER: Elsevier Science B.V.
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The inhibitory effects of β- **glucan** (βG), one of the biol. response modifiers, on the induction of chromosomal aberrations in the bone marrow and spermatogonial cells of mice treated with various anti-neoplastic drugs were investigated. β- **Glucan** (100 mg/kg bw, i.p.) pre-treatment reduced the total number of cells with structural chromosomal aberrations scored after the treatment with **cyclophosphamide** (CP) (2.5 mg/kg bw, i.p.) adriamycin (ADR) (12 mg/kg bw, i.p.) and cis-diamminedichloroplatinum-II (cisplatin) (5 mg/kg bw, i.p.) by about 41.1, 26.9 and 57.7% in bone marrow and 44.4, 55 and 57.1% in spermatogonial cells, resp. This protective effect of β- **glucan** could be attributed to its scavenging ability to trap free-radicals produced during the biotransformation of these anti-neoplastic drugs. β- **Glucan** also markedly restored the mitotic activity of bone marrow cells that had been suppressed by the anti-neoplastic drugs. These results indicate that in addition to the known immunopotentiating activity of β- **glucan**, it plays a role in reducing genotoxicity induced by anti-neoplastic drugs during **cancer** chemotherapy.
REFERENCE COUNT: 48 THERE ARE 48 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 4 OF 9 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2000:324986 CAPLUS
DOCUMENT NUMBER: 133:202741
TITLE: Induction of apoptosis in human prostatic cancer cells with β-glucan (Maitake mushroom polysaccharide)
AUTHOR(S): Fullerton, Sean A.; Samadi, Albert A.; Tortorelis, Dean G.; Choudhury, Muhammad S.; Mallouh, Camille; Tazaki, Hiroshi; Konno, Sensuke
CORPORATE SOURCE: Department of Urology, New York Medical College, Valhalla, NY, USA
SOURCE: Molecular Urology (2000), 4(1), 7-13
CODEN: MOURFE; ISSN: 1091-5362
PUBLISHER: Mary Ann Liebert, Inc.
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Human prostate cancer PC-3 cells were treated with various concns. of the highly purified β-glucan preparation Grifron-D (GD), and viability was determined after 24 h. Lipid peroxidn. (LPO) assay and in situ hybridization (ISH) were performed to evaluate the antitumor mechanism of GD. A concentration-response study showed that almost complete (>95%) cell death was attained in 24 h with GD ≥480 μg/mL. Combinations of GD in a

concentration as low as 30-60 μg/mL with 200 μM vitamin C were as effective as GD alone at 480 μg/mL, inducing >90% cytotoxic cell death. Simultaneous use with various anticancer drugs showed little potentiation of their efficacy, except for the carmustine/GD combination (.apprx.90% reduction in cell viability). The 2-fold elevated LPO level and pos. ISH staining of GD-treated cells indicated oxidative membrane damage resulting in apoptotic cell death. Thus, a bioactive β-glucan from the Maitake mushroom has a cytotoxic effect, presumably through oxidative stress, on prostatic cancer cells in vitro, leading to apoptosis. Potentiation of GD action by vitamin C and the chemosensitizing effect of GD on carmustine may also have clin. implications. This unique mushroom polysaccharide may have potential as an alternative therapeutic modality for prostate cancer.

REFERENCE COUNT: 26 THERE ARE 26 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 5 OF 9 CAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 1998:803767 CAPLUS

DOCUMENT NUMBER: 130:204804

TITLE: In vitro and in vivo hematopoietic activities of Betafectin PGG-glucan

AUTHOR(S): Patchen, Myra L.; Vaudrain, Tracy; Correira, Heidi; Martin, Tracey; Reese, Debrah

CORPORATE SOURCE: Alpha-Beta Technology, Worcester, MA, USA

SOURCE: Experimental Hematology (Charlottesville, Virginia) (1998), 26(13), 1247-1254
CODEN: EXHMA6; ISSN: 0301-472X

PUBLISHER: Carden Jennings Publishing

DOCUMENT TYPE: Journal

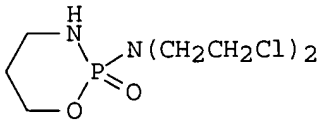
LANGUAGE: English

AB Betafectin PGG-glucan is a novel β-(1,3)glucan that has broad-spectrum anti-infective activities without cytokine induction. Here the authors report that PGG-glucan also has both in vitro and in vivo hematopoietic activities. In vitro studies with bone marrow target cells from the C3H/HeN mouse revealed that although PGG-glucan alone had no direct effect on hematopoietic colony-forming cell (CFC) growth, when combined with granulocyte colony-stimulating factor (CSF) or granulocyte-macrophage CSF, it increased CFC nos. 1.5- to 2.0-fold over those obtained with CSFs alone. Bone marrow cells cultured for high-proliferative-potential CFCs in the presence of interleukin (IL)-1, IL-3, macrophage CSF, and stem cell factor (SCF), or cultured for erythroid burst-forming units in the presence of IL-3, SCF, and erythropoietin, also exhibited enhanced growth in the presence of PGG-glucan. The synergistic effect of PGG-glucan was specific and could be abrogated by anti-PGG-glucan antibody. The ability of PGG-glucan to modulate hematopoiesis in vivo was evaluated in myelosuppressed rodents and primates. C3H/HeN female mice were i.v. administered saline solution or PGG-glucan (0.5 mg/kg) 24 h before the i.p. administration of cyclophosphamide (200 mg/kg), and the recovery of bone marrow cellularity and granulocyte-macrophage progenitor cells was evaluated on days 4 and 8 after cyclophosphamide treatment. At both time points, enhanced hematopoietic recovery was observed in PGG-glucan-treated mice compared with saline-treated control mice. In a final series of in vivo expts., the authors evaluated the ability of therapeutically administered PGG-glucan to enhance hematopoietic recovery in cyclophosphamide-treated cynomolgus monkeys. Monkeys received i.v. infusions of cyclophosphamide (55 mg/kg) on days 1 and 2, followed on days 3 and 10 by i.v. infusion of PGG-glucan (0.5, 1.0, or 2.0 mg/kg). Compared with those in saline-treated monkeys, accelerated white blood cell recovery and a reduction in the median duration of neutropenia were observed in PGG-glucan-treated monkeys. These studies illustrate that PGG-glucan has both in vitro and in vivo hematopoietic activities and that this agent may be useful in the prevention and/or treatment of chemotherapy-associated myelosuppression.

REFERENCE COUNT: 45 THERE ARE 45 CITED REFERENCES AVAILABLE FOR THIS

RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

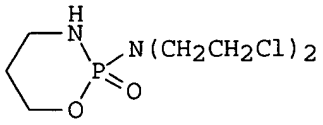
L3 ANSWER 6 OF 9 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1978:453416 CAPLUS
DOCUMENT NUMBER: 89:53416
TITLE: Enhancement of the inhibitory effect of cyclophosphamide on experimental acute myelogenous leukemia by glucan immunopotentiation and response of serum lysozyme
AUTHOR(S): Di Luzio, N. R.; Cook, J. A.; Cohen, C.; Rodrigue, J.; Kokoshis, P.; McNamee, R. B.
CORPORATE SOURCE: Dep. Physiol., Tulane Univ. Sch. Med., New Orleans, LA, USA
SOURCE: Progress in Cancer Research and Therapy (1978), 7(Immune Modulation Control Neoplasia Adjuvant Ther.), 171-82
CODEN: PCRTDK; ISSN: 0145-3726
DOCUMENT TYPE: Journal
LANGUAGE: English
GI



I

AB Tumor growth was reduced in rats receiving either **cyclophosphamide** (I) [50-18-0] or **glucan** [9012-72-0] alone compared to control rats. The most effective antineoplastic action, however, was evident with concurrent **glucan** and I therapy as denoted by a mean 97% decrease in tumor weight compared to control rats. Phagocytic activity of the reticuloendothelial system was subsequently evaluated after singular or combined administration of **glucan** and I. I abrogated the **glucan**-induced hyperphagocytic state even though interaction of these 2 agents was extremely effective in inducing tumor regression. Increased survival to i.v. administered acute myelogenous leukemic cells was also observed in the **glucan**- and I-treated group. I inhibited **glucan**-induced hepatic and pulmonary granuloma. **Glucan** elevated serum lysozyme [9001-63-2] concns. in both the presence and absence of I. **Glucan** may be a valuable adjunct to conventional **cancer** chemotherapy.

L3 ANSWER 7 OF 9 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1978:453298 CAPLUS
DOCUMENT NUMBER: 89:53298
TITLE: The synergistic effect of cyclophosphamide and glucan on experimental acute myelogenous and lymphocytic leukemia
AUTHOR(S): Di Luzio, N. R.; Cook, J. A.; Cohen, C.; Rodrigue, J.; Jones, E.
CORPORATE SOURCE: Dep. Physiol., Tulane Univ. Sch. Med., New Orleans, LA, USA
SOURCE: Proc. EURES Symp. Macrophage Cancer (1977), 188-201. Editor(s): James, Keith; McBride, Bill; Stuart, Angus. Univ. Edinburgh, Med. Sch.: Edinburgh, Scot.
CODEN: 38BZA9
DOCUMENT TYPE: Conference
LANGUAGE: English
GI



I

AB In rats with Shay myelogenous leukemia, primary tumor growth was significantly reduced after administration of either **cyclophosphamide** (I) [50-18-0] (40 mg/kg, i.p., on days 3 and 6) or **glucan** [9012-72-0] (10 mg/kg, i.v. on days 3 and 6) alone compared to control rats. The most effective antineoplastic action, however, was evident with concurrent **glucan** and I therapy as denoted by a mean 97% decrease in tumor weight compared to control rats. In mice, increased survival after i.v. administered acute myelogenous leukemic cells was also observed in the **glucan** and I-treated group. I inhibited, to some degree, the **glucan**-induced hepatic granuloma. The degree of hepatic metastases was significantly reduced in both rats and mice by the conjoint use of I and **glucan**. Thus, **glucan** may be a valuable adjunct to conventional **cancer** chemotherapy.

L3 ANSWER 8 OF 9 MEDLINE on STN
ACCESSION NUMBER: 2005643622 IN-PROCESS
DOCUMENT NUMBER: PubMed ID: 16327179
TITLE: Soy isoflavone aglycone modulates a hematopoietic response in combination with soluble beta-glucan: SCG.
AUTHOR: Harada Toshie; Masuda Susumu; Arii Masayuki; Adachi Yoshiyuki; Nakajima Mitsuhiro; Yadomae Toshiro; Ohno Naohito
CORPORATE SOURCE: Laboratory for Immunopharmacology of Microbial Products, School of Pharmacy, Tokyo University of Pharmacy & Life Science, Hachioji, Japan.
SOURCE: Biological & pharmaceutical bulletin, (2005 Dec) 28 (12) 2342-5.
Journal code: 9311984. ISSN: 0918-6158.
PUB. COUNTRY: Japan
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: NONMEDLINE; IN-PROCESS; NONINDEXED; Priority Journals
ENTRY DATE: Entered STN: 20051206
Last Updated on STN: 20051221

AB Soy isoflavone aglycones (IFAs) have a wide range of biological actions that suggest they may be of use in **cancer** prevention. On the other hand, a branched beta-**glucan** from Sparassis crispa (SCG) is a major 6-branched 1,3-beta-D-**glucan** in an edible/medicinal mushroom: Sparassis crispa showing antitumor activity. We have previously reported that both oral and intraperitoneal administration of SCG enhanced the hematopoietic response in **cyclophosphamide** (CY)-induced leukopenic mice. In this study, we investigated the hematopoietic response due to IFA in combination with SCG in CY-induced leukopenic mice. The oral administration of IFA in combination with SCG synergistically enhanced the number of white blood cells, and increased spleen weight. Analyzing the leukocyte population by flow cytometry, the combination of IFA and SCG increased the number of monocytes and granulocytes in the spleen. Taken together, the combination of IFA and SCG synergistically provides the hematopoietic responses that are enhanced over IFA or SCG alone.

L3 ANSWER 9 OF 9 MEDLINE on STN
ACCESSION NUMBER: 2003491804 MEDLINE
DOCUMENT NUMBER: PubMed ID: 14568293

TITLE: Beta-glucan inhibits the genotoxicity of cyclophosphamide, adriamycin and cisplatin.

AUTHOR: Tohamy Amany A; El-Ghor Akmal A; El-Nahas Soheir M; Noshy Magda M

CORPORATE SOURCE: Zoology Department, Faculty of Science, Helwan University, Cairo, Egypt.

SOURCE: Mutation research, (2003 Nov 10) 541 (1-2) 45-53.
Journal code: 0400763. ISSN: 0027-5107.

PUB. COUNTRY: Netherlands

DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)

LANGUAGE: English

FILE SEGMENT: Priority Journals

ENTRY MONTH: 200312

ENTRY DATE: Entered STN: 20031022
Last Updated on STN: 20031219
Entered Medline: 20031203

AB The inhibitory effects of beta-**glucan** (betaG), one of the biological response modifiers, on the induction of chromosomal aberrations in the bone marrow and spermatogonial cells of mice treated with various anti-neoplastic drugs were investigated. beta-**Glucan** (100 mg/kg bw, i.p.) pre-treatment reduced the total number of cells with structural chromosomal aberrations scored after the treatment with **cyclophosphamide** (CP) (2.5 mg/kg bw, i.p.) adriamycin (ADR) (12 mg/kg bw, i.p.) and cis-diamminedichloroplatinum-II (cisplatin) (5 mg/kg bw, i.p.) by about 41.1, 26.9 and 57.7% in bone marrow and 44.4, 55 and 57.1% in spermatogonial cells, respectively. This protective effect of beta-**glucan** could be attributed to its scavenging ability to trap free-radicals produced during the biotransformation of these anti-neoplastic drugs. Beta-**glucan** also markedly restored the mitotic activity of bone marrow cells that had been suppressed by the anti-neoplastic drugs. These results indicate that in addition to the known immunopotentiating activity of beta-**glucan**, it plays a role in reducing genotoxicity induced by anti-neoplastic drugs during **cancer** chemotherapy.

L5 ANSWER 1 OF 1 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1992:503739 CAPLUS
DOCUMENT NUMBER: 117:103739
TITLE: Suppressing effects of glucan on micronuclei induced by cyclophosphamide in mice
AUTHOR(S): Chorvatovicova, Darina; Navarova, Jana
CORPORATE SOURCE: Inst. Ecobiol., Slovak Acad. Sci., Bratislava, 814 34, Czech.
SOURCE: Mutation Research (1992), 282(3), 147-50
CODEN: MUREAV; ISSN: 0027-5107
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The effect of pretreatment with **carboxymethylglucan** (CMG) on the frequency of micronuclei induced by **cyclophosphamide** administration in mice was evaluated. Two doses of CMG (50 mg/kg) injected either i.p. 24 h or i.v. 1 h prior to two **cyclophosphamide** administrations (80 mg/kg) significantly decreased the frequency of micronucleated PCE in bone marrow. Of two evaluated derivs. of **carboxymethylglucan**, the K3 derivative was most efficient. The results show that it is possible to achieve a suppressive effect of soluble **carboxymethylglucan** prepared from Saccharomyces cerevisiae against **cyclophosphamide** mutagenicity. The notion may be useful for **glucan**'s effects against pharmacocarcinogenesis. Therapeutic application of **glucan** with **cyclophosphamide** therapy may provide a remarkable decrease of the secondary **tumor** risk. The utilization of these results for human **patients** needs to be considered.

> d l13 1-2 ibib abs

L13 ANSWER 1 OF 2 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2005:394530 CAPLUS
DOCUMENT NUMBER: 142:423818
TITLE: Therapeutical combination against cancer comprising a monoclonal antibody with a glucan
INVENTOR(S): Yvin, Jean-Claude; Panak, Edouard; Vetvicka, Vaclav
PATENT ASSIGNEE(S): Fr.
SOURCE: U.S. Pat. Appl. Publ., 6 pp.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2005095250	A1	20050505	US 2003-698034	20031030
WO 2005049044	A1	20050602	WO 2004-EP13119	20041029

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.: US 2003-698034 A 20031030

AB The present invention relates to compns. and methods for treating humans and warm-blood animals suffering from **cancer**. More particularly, a therapeutical treatment in which a monoclonal antibody is administered with either β-(1,3)-glucan like **laminarin** or an oligo-β-(1,3)-glucan and a pharmaceutically acceptable carrier, to patients suffering from **cancer** are described. Female nude mice were implanted s.c. with human breast carcinoma cell line. Mice were injected i.p. with combination of Phycarine 500 mg/kg, once a day for 5 days and Herceptin 0.5 mg/kg, twice a week during 3 wk. The combined administration of Phycarine and Herceptin allowed a limitation in the increase of the **tumor** weight which was far higher than the mean value obtained when administering Herceptin or Phycarine alone; said activity on the **tumor** weight being even equivalent to the one obtained when administering a conventional dosage of taxol.

L13 ANSWER 2 OF 2 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2003:434382 CAPLUS
DOCUMENT NUMBER: 139:12302
TITLE: Laminaria polysaccharides for therapeutical treatments
INVENTOR(S): Yvin, Jean-Claude; Vetvicka, Vaclav
PATENT ASSIGNEE(S): Laboratoires Goeemar S.A., Fr.
SOURCE: PCT Int. Appl., 29 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2003045414	A2	20030605	WO 2002-EP13512	20021129

WO 2003045414 A3 20031016
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SC, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZW
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

US 2003119780	A1	20030626	US 2001-999202	20011130
US 6660722	B2	20031209		
CA 2468314	AA	20030605	CA 2002-2468314	20021129
AU 2002352187	A1	20030610	AU 2002-352187	20021129
EP 1448215	A2	20040825	EP 2002-787872	20021129
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, SK				
JP 2005510543	T2	20050421	JP 2003-546915	20021129

PRIORITY APPLN. INFO.: US 2001-999202 A 20011130
WO 2002-EP13512 W 20021129

AB A therapeutical method comprises administration to a patient of an effective amount of especially soluble **laminarin** for the treatment of **tumors** and more generally of **cancers** of the group comprising breast **cancer**, lung **cancer**, esophagus **cancer**, stomach **cancer**, intestine and colon **cancers**, and for the treatment of viral, bacterial and fungal diseases as well as diseases related to immunostimulant deficiencies of human beings and warm-blood animals.

L12 ANSWER 17 OF 18 MEDLINE on STN
ACCESSION NUMBER: 1999426885 MEDLINE
DOCUMENT NUMBER: PubMed ID: 10495437
TITLE: Inhibition of heparanase activity and **tumor** metastasis by **laminarin** sulfate and synthetic phosphorothioate oligodeoxynucleotides.
AUTHOR: Miao H Q; Elkin M; Aingorn E; Ishai-Michaeli R; Stein C A; Vlodavsky I
CORPORATE SOURCE: Department of Oncology, Hadassah University Hospital, Jerusalem, Israel.
SOURCE: International journal of cancer. Journal international du cancer, (1999 Oct 29) 83 (3) 424-31.
Journal code: 0042124. ISSN: 0020-7136.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199910
ENTRY DATE: Entered STN: 19991101
Last Updated on STN: 19991101
Entered Medline: 19991021

AB Heparanase activity correlates with the metastatic potential of **tumor** cells. Moreover, the anti-metastatic effect of non-anti-coagulant species of heparin and certain sulfated polysaccharides was attributed to their heparanase-inhibiting activity. We investigated the effect of a chemically sulfated polysaccharide (**laminarin**), consisting primarily of beta-1,3 glucan (sodium **laminarin**), and of synthetic phosphorothioate oligodeoxynucleotides, primarily phosphorothioate homopolymer of cytidine (SdC28), on heparanase activity and **tumor** metastasis. Investigation of the ability of **tumor** cells to degrade heparan sulfate in intact extracellular matrix revealed that heparanase activity expressed by B16-BL6 mouse melanoma cells and 13762 MAT rat mammary adenocarcinoma cells was effectively inhibited by LS (50% inhibition at 0.2-1 microgram/ml), but there was no inhibition by sodium **laminarin** up to a concentration of 50 microgram/ml. Complete inhibition of the melanoma heparanase was obtained in the presence of 0.1 microM SdC28. A single i.p. injection of **laminarin** sulfate, but not of sodium **laminarin**, before i.v. inoculation of the melanoma or breast-carcinoma cells inhibited the extent of lung colonization by the **tumor** cells by 80 to 90%. Similar inhibition was exerted by 0.1 microM SdC28. At the effective concentrations, both compounds had a small effect on proliferation of the **tumor** cells and on growth of the primary **tumors** in vivo. These results further emphasize the involvement of heparanase in **tumor** metastasis and the potential clinical application of diverse heparanase-inhibiting molecules such as sulfated polysaccharides and synthetic polyanionic molecules.

L12 ANSWER 16 OF 18 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1963:444312 CAPLUS
DOCUMENT NUMBER: 59:44312
ORIGINAL REFERENCE NO.: 59:8030h
TITLE: Effects of sulfated degraded **laminarin** on experimental **tumor** growth
AUTHOR(S): Jolles, B.; Remington, Mary; Andrews, P. S.
CORPORATE SOURCE: Gen. Hosp., Northampton, UK
SOURCE: British Journal of Cancer (1963), 17, 109-15
CODEN: BJCAAI; ISSN: 0007-0920
DOCUMENT TYPE: Journal
LANGUAGE: Unavailable
AB The compound, a polysaccharide derivative, inhibited the growth of sarcoma 180 when injected at the site of the transplant or into growing tumors.

references.

L12 ANSWER 15 OF 18 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1966:441776 CAPLUS
DOCUMENT NUMBER: 65:41776
ORIGINAL REFERENCE NO.: 65:7840d-f
TITLE: Comparative study of the biological action of polysaccharides glucan and laminarin
AUTHOR(S): Fomina, I. P.; Navashin, S. M.; Preobrazhenskaya, M. E.; Rozenfel'd, E. L.
CORPORATE SOURCE: Res. Inst. Antibiots., Moscow
SOURCE: Byulleten Eksperimental'noi Biologii i Meditsiny (1966), 61(5), 79-83
CODEN: BEBMAE; ISSN: 0365-9615
DOCUMENT TYPE: Journal
LANGUAGE: Russian

AB Albino mice were used for comparing biol. activity of glucan and **laminarin**. In the 1st series of expts., the animals were injected with glucan or **laminarin** in doses of 5-30 mg./kg., and then subjected to the action of following microorganisms: [Salmonella typhosa [Bacillus] dysenteriae sonne [Shigella sonne], Escherichia coli, Proteus mirabilis, Pseudomonas aeruginosa, Staph[ylococcus] aureus, S. albus, and D[iplococcus] pneumoniae. Glucan and **laminarin** produced nearly identical preventive effects in exptl. staphylococcic sepsis: the survival of treated animals was 76-83%, whereas the death rate of controls was 90%. In sepsis induced by gram-neg. microorganisms, glucan showed a favorable influence, while **laminarin** proved ineffective. In the 2nd exptl. series, an antitumor effect of both polysaccharides was observed. Glucan, 20 mg./kg. produced a pronounced antitumor effect against Ehrlich **tumor** and sarcoma 180, inhibiting their growth by 53-60%. No inhibitory activity of **laminarin** was found under analogous conditions. Since both polysaccharides were of the same mol. structure, it is suggested that different biol. activities was due to their size and configuration. This explanation is supported by a reduction in glucan biol. activity after splitting off some of its glucose residues.

references.

L12 ANSWER 15 OF 18 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1966:441776 CAPLUS
DOCUMENT NUMBER: 65:41776
ORIGINAL REFERENCE NO.: 65:7840d-f
TITLE: Comparative study of the biological action of polysaccharides glucan and laminarin
AUTHOR(S): Fomina, I. P.; Navashin, S. M.; Preobrazhenskaya, M. E.; Rozenfel'd, E. L.
CORPORATE SOURCE: Res. Inst. Antibiots., Moscow
SOURCE: Byulleten Eksperimental'noi Biologii i Meditsiny (1966), 61(5), 79-83
CODEN: BEBMAE; ISSN: 0365-9615
DOCUMENT TYPE: Journal
LANGUAGE: Russian

AB Albino mice were used for comparing biol. activity of glucan and **laminarin**. In the 1st series of expts., the animals were injected with glucan or **laminarin** in doses of 5-30 mg./kg., and then subjected to the action of following microorganisms: [Salmonella typhosa [Bacillus] dysenteriae sonne [Shigella sonne], Escherichia coli, Proteus mirabilis, Pseudomonas aeruginosa, Staph[ylococcus] aureus, S. albus, and D[iplococcus] pneumoniae. Glucan and **laminarin** produced nearly identical preventive effects in exptl. staphylococcic sepsis: the survival of treated animals was 76-83%, whereas the death rate of controls was 90%. In sepsis induced by gram-neg. microorganisms, glucan showed a favorable influence, while **laminarin** proved ineffective. In the 2nd exptl. series, an antitumor effect of both polysaccharides was observed. Glucan, 20 mg./kg. produced a pronounced antitumor effect against Ehrlich **tumor** and sarcoma 180, inhibiting their growth by 53-60%. No inhibitory activity of **laminarin** was found under analogous conditions. Since both polysaccharides were of the same mol. structure, it is suggested that different biol. activities was due to their size and configuration. This explanation is supported by a reduction in glucan biol. activity after splitting off some of its glucose residues.

references.

L12 ANSWER 15 OF 18 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1966:441776 CAPLUS
DOCUMENT NUMBER: 65:41776
ORIGINAL REFERENCE NO.: 65:7840d-f
TITLE: Comparative study of the biological action of polysaccharides glucan and laminarin
AUTHOR(S): Fomina, I. P.; Navashin, S. M.; Preobrazhenskaya, M. E.; Rozenfel'd, E. L.
CORPORATE SOURCE: Res. Inst. Antibiots., Moscow
SOURCE: Byulleten Eksperimental'noi Biologii i Meditsiny (1966), 61(5), 79-83
CODEN: BEBMAE; ISSN: 0365-9615
DOCUMENT TYPE: Journal
LANGUAGE: Russian

AB Albino mice were used for comparing biol. activity of glucan and **laminarin**. In the 1st series of expts., the animals were injected with glucan or **laminarin** in doses of 5-30 mg./kg., and then subjected to the action of following microorganisms: [Salmonella typhosa [Bacillus] dysenteriae sonne [Shigella sonne], Escherichia coli, Proteus mirabilis, Pseudomonas aeruginosa, Staph[ylococcus] aureus, S. albus, and D[iplococcus] pneumoniae. Glucan and **laminarin** produced nearly identical preventive effects in exptl. staphylococcic sepsis: the survival of treated animals was 76-83%, whereas the death rate of controls was 90%. In sepsis induced by gram-neg. microorganisms, glucan showed a favorable influence, while **laminarin** proved ineffective. In the 2nd exptl. series, an antitumor effect of both polysaccharides was observed. Glucan, 20 mg./kg. produced a pronounced antitumor effect against Ehrlich **tumor** and sarcoma 180, inhibiting their growth by 53-60%. No inhibitory activity of **laminarin** was found under analogous conditions. Since both polysaccharides were of the same mol. structure, it is suggested that different biol. activities was due to their size and configuration. This explanation is supported by a reduction in glucan biol. activity after splitting off some of its glucose residues.

L12 ANSWER 15 OF 18 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1966:441776 CAPLUS
DOCUMENT NUMBER: 65:41776
ORIGINAL REFERENCE NO.: 65:7840d-f
TITLE: Comparative study of the biological action of polysaccharides glucan and laminarin
AUTHOR(S): Fomina, I. P.; Navashin, S. M.; Preobrazhenskaya, M. E.; Rozenfel'd, E. L.
CORPORATE SOURCE: Res. Inst. Antibiots., Moscow
SOURCE: Byulleten Eksperimental'noi Biologii i Meditsiny (1966), 61(5), 79-83
CODEN: BEBMAE; ISSN: 0365-9615
DOCUMENT TYPE: Journal
LANGUAGE: Russian

AB Albino mice were used for comparing biol. activity of glucan and **laminarin**. In the 1st series of expts., the animals were injected with glucan or **laminarin** in doses of 5-30 mg./kg., and then subjected to the action of following microorganisms: [Salmonella typhosa [Bacillus] dysenteriae sonne [Shigella sonne], Escherichia coli, Proteus mirabilis, Pseudomonas aeruginosa, Staph[ylococcus] aureus, S. albus, and D[iplococcus] pneumoniae. Glucan and **laminarin** produced nearly identical preventive effects in exptl. staphylococcic sepsis: the survival of treated animals was 76-83%, whereas the death rate of controls was 90%. In sepsis induced by gram-neg. microorganisms, glucan showed a favorable influence, while **laminarin** proved ineffective. In the 2nd exptl. series, an antitumor effect of both polysaccharides was observed. Glucan, 20 mg./kg. produced a pronounced antitumor effect against Ehrlich **tumor** and sarcoma 180, inhibiting their growth by 53-60%. No inhibitory activity of **laminarin** was found under analogous conditions. Since both polysaccharides were of the same mol. structure, it is suggested that different biol. activities was due to their size and configuration. This explanation is supported by a reduction in glucan biol. activity after splitting off some of its glucose residues.

L12 ANSWER 11 OF 18 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1996:423114 CAPLUS
DOCUMENT NUMBER: 125:131856
TITLE: Inhibition of angiogenesis and murine **tumor** growth by **laminarin** sulfate
AUTHOR(S): Hoffman, R.; Paper, D. H.; Donaldson, J.; Vogl, H.
CORPORATE SOURCE: Clinical Oncology and Radiotherapeutics Unit, MRC Centre, Cambridge, CB2 2QH, UK
SOURCE: British Journal of Cancer (1996), 73(10), 1183-1186
CODEN: BJCAAI; ISSN: 0007-0920
PUBLISHER: Stockton
DOCUMENT TYPE: Journal
LANGUAGE: English
AB LAM S5 is a polysulfated derivative of the glucan **laminarin** that inhibits basic fibroblast growth factor (bFGF) binding and the bFGF-stimulated proliferation of fetal bovine heart endothelial (FBHE) cells. This report demonstrates that LAM S5 has anti-angiogenic activity, as shown by inhibition of tubule formation by endothelial cells cultured on Matrigel and inhibition of vascularization of the chick chorioallantoic membrane. In addition, LAM S5 caused a **tumor** growth delay of the murine RIF-1 **tumor** of 2.6 days.

L12 ANSWER 1 OF 18 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2005:394530 CAPLUS
DOCUMENT NUMBER: 142:423818
TITLE: Therapeutical combination against cancer comprising a monoclonal antibody with a glucan
INVENTOR(S): Yvin, Jean-Claude; Panak, Edouard; Vetvicka, Vaclav
PATENT ASSIGNEE(S): Fr.
SOURCE: U.S. Pat. Appl. Publ., 6 pp.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2005095250	A1	20050505	US 2003-698034	20031030
WO 2005049044	A1	20050602	WO 2004-EP13119	20041029

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.: US 2003-698034 A 20031030
AB The present invention relates to compns. and methods for treating humans and warm-blood animals suffering from cancer. More particularly, a therapeutical treatment in which a monoclonal antibody is administered with either β-(1,3)-glucan like **laminarin** or an oligo-β-(1,3)-glucan and a pharmaceutically acceptable carrier, to patients suffering from cancer are described. Female nude mice were implanted s.c. with human breast carcinoma cell line. Mice were injected i.p. with combination of Phycarine 500 mg/kg, once a day for 5 days and Herceptin 0.5 mg/kg, twice a week during 3 wk. The combined administration of Phycarine and Herceptin allowed a limitation in the increase of the **tumor** weight which was far higher than the mean value obtained when administering Herceptin or Phycarine alone; said activity on the **tumor** weight being even equivalent to the one obtained when administering a conventional dosage of taxol.

L12 ANSWER 2 OF 18 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2004:58418 CAPLUS
DOCUMENT NUMBER: 141:386
TITLE: Role of selenium in antioxidative effect of heparin-selenocystamine
AUTHOR(S): Saito, Yoshihiro; Tsuda, Tsubasa; Eguchi, Ryoko; Sato, Takaji; Chikuma, Masahiko
CORPORATE SOURCE: Department of Bio-analytical Chemistry, Osaka University of Pharmaceutical Sciences, Osaka, 569-1094, Japan
SOURCE: Biomedical Research on Trace Elements (2003), 14(4), 329-331
CODEN: BRTEE5; ISSN: 0916-717X
PUBLISHER: Nippon Biryo Genso Gakkai
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Heparin-cystamine (Hep-Cyst), **laminarin**-selenocystamine

(Lam-SeCyst), and fucoidan-selenocystamine (Fuc-SeCyst) conjugates were newly synthesized by the same method as that for heparin-selenocystamine (Hep-SeCyst) which we have prepared before. Antioxidative effects of the selenocystamine (SeCyst) conjugates were compared with those of Hep-Cyst to clarify the role of selenium in SeCyst conjugates. Hep-Cyst had thiol groups in the mol., while SeCyst conjugates had selenol groups. At pH 6.0, Hep-SeCyst reacted with DTNB, but Hep-Cyst did not, though both of the conjugates reacted with DTNB at pH 8.0. It is considered that the result is caused by the difference in pKa value of thiol and selenol groups in the conjugates. Both Hep-SeCyst and Hep-Cyst had DPPH radical scavenging activity, and Hep-SeCyst showed higher activity than Hep-Cyst. The viability of Ehrlich ascites **tumor** cells (EATC), which was decreased by DPPH treatment, recovered by the simultaneous addition of SeCyst or Cyst conjugates, indicating that these conjugates have protective effect on EATC from oxidative damages induced by DPPH. The cytoprotective effects of SeCyst conjugates were also higher than that of Hep-Cyst. These results suggested that higher reactivity of selenol groups in SeCyst conjugates may be a primary factor of higher antioxidative activities, i.e., DPPH scavenging activity and cytoprotective activity against DPPH-induced oxidative damage.

REFERENCE COUNT: 3 THERE ARE 3 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L12 ANSWER 3 OF 18 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2003:434382 CAPLUS
DOCUMENT NUMBER: 139:12302
TITLE: Laminaria polysaccharides for therapeutical treatments
INVENTOR(S): Yvin, Jean-Claude; Vetvicka, Vaclav
PATENT ASSIGNEE(S): Laboratoires Goeemar S.A., Fr.
SOURCE: PCT Int. Appl., 29 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2003045414	A2	20030605	WO 2002-EP13512	20021129
WO 2003045414	A3	20031016		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SC, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZW				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
US 2003119780	A1	20030626	US 2001-999202	20011130
US 6660722	B2	20031209		
CA 2468314	AA	20030605	CA 2002-2468314	20021129
AU 2002352187	A1	20030610	AU 2002-352187	20021129
EP 1448215	A2	20040825	EP 2002-787872	20021129
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, SK				
JP 2005510543	T2	20050421	JP 2003-546915	20021129

PRIORITY APPLN. INFO.: US 2001-999202 A 20011130
WO 2002-EP13512 W 20021129

AB A therapeutical method comprises administration to a patient of an effective amount of especially soluble **laminarin** for the treatment of **tumors** and more generally of cancers of the group comprising

breast cancer, lung cancer, esophagus cancer, stomach cancer, intestine and colon cancers, and for the treatment of viral, bacterial and fungal diseases as well as diseases related to immunostimulant deficiencies of human beings and warm-blood animals.

L12 ANSWER 4 OF 18 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2003:324193 CAPLUS
DOCUMENT NUMBER: 139:345597
TITLE: Study on mechanism of laminarin sulfate in prevention of experimental atherosclerosis
AUTHOR(S): Liang, Xuguo; Du, Xiaoxia; Pan, Qixing
CORPORATE SOURCE: Department of Cardiology, Qilu Hospital, Shangdong University, Jinan, 250012, Peop. Rep. China
SOURCE: Zhongguo Haiyang Yaowu (2002), 21(5), 26-30
CODEN: ZHYAE8; ISSN: 1002-3461
PUBLISHER: Shandongsheng Haiyang Yaowu Kexue Yanjiuso
DOCUMENT TYPE: Journal
LANGUAGE: Chinese
AB The possible immunol. mechanism of **laminarin** sulfate in the prevention of exptl. atherosclerosis was analyzed. Serum soluble interleukin 2 receptor (sIL-2R), circulating immuno-complex, subunits of T lymphocyte, interleukin-6 (IL-6), interleukin-8 (IL-8), **tumor** necrosis factor-α (TNF-α) and lipid metabolism were determined by ELISA, RIA in rats and quails. The lipid metabolism and immunol. function were prominently disturbed in animals after feeding with high-lipid food. **Laminarin** sulfate showed obvious regulating effects on above-mentioned index. The mechanism of **laminarin** sulfate in the prevention of atherosclerosis might be closely related to the regulation of the disturbance of lipid metabolism and to the regulation of the immunol. function of the body.

L12 ANSWER 5 OF 18 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2003:321189 CAPLUS
DOCUMENT NUMBER: 139:51655
TITLE: Induction of TNF-α production from human peripheral blood monocytes with β-1,3-glucan oligomer prepared from laminarin with β-1,3-glucanase from Bacillus clausii NM-1
AUTHOR(S): Miyanishi, Nobumitsu; Iwamoto, Yoshiko; Watanabe, Etsuo; Oda, Tatsuya
CORPORATE SOURCE: Department of Food Science and Technology, Tokyo University of Fisheries, Tokyo, 108-8477, Japan
SOURCE: Journal of Bioscience and Bioengineering (2003), 95(2), 192-195
CODEN: JBBIF6; ISSN: 1389-1723
PUBLISHER: Society for Bioscience and Bioengineering, Japan
DOCUMENT TYPE: Journal
LANGUAGE: English
AB We prepared a β-1,3-glucan oligomer (DP≥4) from laminarin (DP: 25-30) derived from Laminaria digitata with β-1,3-glucanase, and examined its effect on human peripheral blood monocytes. Conditioned medium prepared by incubating monocytes (MC-CM) with the β-1,3-glucan oligomer showed strong inhibitory activity against the proliferation of human leukemic U937 cells. Since the β-1,3-glucan oligomer had no direct cytotoxic effect on U937 cells up to 1000 μg/mL, the cytotoxicity of the MC-CM may be due to cytotoxic cytokines produced from monocytes stimulated by the β-1,3-glucan oligomer. On the other hand, the MC-CM prepared with original laminarin had little effect on the growth of U937 cells. The cytotoxicity of the MC-CM prepared with the β-1,3-glucan oligomer was significantly reduced by an anti-TNF-α antibody, but the anti-TNF-β antibody had no effect. Our results suggest that the enzymically depolymd. β-1,3-glucan oligomer induces TNF-α production from human monocytes.

REFERENCE COUNT: 17 THERE ARE 17 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L12 ANSWER 6 OF 18 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2003:319914 CAPLUS
DOCUMENT NUMBER: 138:304468
TITLE: Method of preparing purified biologically active laminarin oligosaccharide libraries
INVENTOR(S): Gulko, Mirit Kolog; Kelson, Idil Kasuto; Grosz-Moraga, Ana; Samokovlisky, Albena; Amor, Yehudit; Markman, Ofer; Shvartser, Leonid
PATENT ASSIGNEE(S): Procognia, Ltd., Israel
SOURCE: PCT Int. Appl., 70 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2003033512	A2	20030424	WO 2002-IB4631	20021016
WO 2003033512	C2	20031030		
WO 2003033512	A3	20031224		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.: US 2001-329744P P 20011016

AB Disclosed are methods of making **laminarin** oligosaccharide libraries whose members have defined structural and/or functional properties, as well as methods of making and using the **laminarin** oligosaccharide libraries. A protein binding profile of various LS fractions was generated by determining the binding affinity of various fractions to a panel of proteins known to bind oligosaccharide mols. The proteins used included fibroblast growth factor (FGF); antithrombin III (ATIII); epidermal growth factor (EGF); interferon (IFN); insulin-like growth factor (IFN); keratinocyte growth factor (KGF); vascular endothelial growth factor (VEGF); Apolipoprotein E4 (ApoE4); hepatocyte growth factor (HGF); and **tumor** necrosis factor (TNF).

L12 ANSWER 7 OF 18 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2000:846470 CAPLUS
DOCUMENT NUMBER: 134:172678
TITLE: Synthesis and heparin-like biological activity of amino acid-based polymers
AUTHOR(S): Bentolila, Alfonso; Vlodavsky, Israel; Haloun, Christine; Domb, Abraham J.
CORPORATE SOURCE: Departments of Medicinal Chemistry, School of Pharmacy-Faculty of Medicine, The Hebrew University of Jerusalem, Jerusalem, 91120, Israel
SOURCE: Polymers for Advanced Technologies (2000), 11(8-12), 377-387
CODEN: PADTE5; ISSN: 1042-7147
PUBLISHER: John Wiley & Sons Ltd.
DOCUMENT TYPE: Journal

LANGUAGE: English
OTHER SOURCE(S): CASREACT 134:172678

AB Biol. macromols. are important regulators of physiol. functions. Most of the biol. active macromols. are charged linear polymers like some proteins, DNA and glycosaminoglycans (GAG). Heparin, the first GAG applied in medicine, is a natural polyanion composed of repeating disaccharide units of glucosamine and uronic acid. The amino and hydroxyl groups of the glucosamine units are partially sulfated. Heparin is a potent anticoagulant, and is also active as an antimethastatic and antiproliferative agent. Sulfatation of other polysaccharides such as **laminarin** yielded very potent new anticoagulants. It was hypothesized that macromols. based on N-acryl L-amino acids bearing hydrophobic or charged side groups, such as -NH2, - COOH, -SH, -OH and phenols, arranged into a configuration determined by the chirality of the amino acid α-carbon, may express heparin-like biol. activities. Homo-poly(N-acryl amino acids) were synthesized from the corresponding monomers. Polymers with different charge densities, nature of the amino acid side group, stereoselectivity and polymeric backbone were tested for their activity as anticoagulants, heparanase inhibition agents, and to basic fibroblast growth factor (b-FGF) release agents bound to the extracellular matrix (ECM). The type of amino acid, the polymer backbone, the charge d. and distribution strongly affect the biol. activity exerted by these polyanions. All polymers being active either as heparanase inhibitors and/or as b-FGF release agents have at least a neg. charge d. of 1 per amino acid residue. Polymers bearing hydrophilic side chains that inhibited heparanase, i.e., hydroxyproline, glycine and serine, did not release b-FGF from ECM. The absence of high acidic sulfate-ester groups existing in heparin (hydrophilic) must be compensated by some kind of lipophilic interactions between the polyanion and b-FGF in order to effectively compete with heparan sulfate proteoglycanes, causing its release from ECM. Heparanase inhibitors may have clin. applications in preventing **tumor** metastasis and inflammatory/autoimmune processes due to the involvement of this enzyme in the extravasation of blood-borne **tumor** cells and activated cells of the immune system. Mols. that release ECM-bound b-FGF may be applied to accelerate neovascularization and tissue repair.

REFERENCE COUNT: 33 THERE ARE 33 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L12 ANSWER 8 OF 18 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1999:773732 CAPLUS
DOCUMENT NUMBER: 132:288446
TITLE: Activation of murine peritoneal macrophages by laminarin
AUTHOR(S): Xue, Jingbo; Liu, Xiying; Zhang, Hongfen
CORPORATE SOURCE: Medical College, Qingdao University, Tsingtao, 266021, Peop. Rep. China
SOURCE: Zhongguo Haiyang Yaowu (1999), 18(3), 23-25
CODEN: ZHYAE8; ISSN: 1002-3461
PUBLISHER: Shandongsheng Haiyang Yaowu Kexue Yanjiuso
DOCUMENT TYPE: Journal
LANGUAGE: Chinese

AB Activation of murine peritoneal macrophages by laminarin was studied in G57BL/6 mice. Peritoneal macrophages could be markedly activated by i.p. injection of laminarin (40 mg/kg) for cytolysis. Laminarin activated peritoneal macrophages secretion of TNF in vitro in the presence of LPS (10 ng/mL).

```
=> d his

     (FILE 'HOME' ENTERED AT 13:02:09 ON 07 FEB 2006)

     FILE 'CAPLUS, MEDLINE' ENTERED AT 13:02:21 ON 07 FEB 2006
L1            83 S ?GLUCAN (P) CYCLOPHOSPHAMIDE
L2            32 S ?GLUCAN (P) CYCLOPHOSPHAMIDE (P) TUMOR?
L3             9 S ?GLUCAN (P) CYCLOPHOSPHAMIDE (P) CANCER?
L4            30 S L2 NOT L3
L5             1 S L4 AND PATIENT?
L6            29 S L4 NOT L5
L7             0 S L6 AND LAMINARIN?
L8             1 S L1 AND LAMINARIN?
L9             1 S LAMINARIN? (P) CYCLOPHOSPHAMIDE (P) CANCER?
L10            0 S LAMINARIN? (P) CYCLOPHOSPHAMIDE (P) TUMOR?
L11            2 S LAMINARIN? (P) CYCLOPHOSPHAMIDE
L12           18 S LAMINARIN (P) TUMOR?
L13            2 S LAMINARIN (P) TUMOR? (P) CANCER?
L14            5 S LAMINARIN (P) CANCER?
L15            3 S LAMINARIN (P) TUMOUR?
L16            1 S LAMINARIN (P) ANTINEOPLASTIC?
L17            1 S LAMINARIN (P) ANTINEOPLAS?
L18            1 S LAMINARIN (P) CHEMOTHERAP?
```

```
=> d his

     (FILE 'HOME' ENTERED AT 13:02:09 ON 07 FEB 2006)

     FILE 'CAPLUS, MEDLINE' ENTERED AT 13:02:21 ON 07 FEB 2006
L1            83 S ?GLUCAN (P) CYCLOPHOSPHAMIDE
L2            32 S ?GLUCAN (P) CYCLOPHOSPHAMIDE (P) TUMOR?
L3             9 S ?GLUCAN (P) CYCLOPHOSPHAMIDE (P) CANCER?
L4            30 S L2 NOT L3
L5             1 S L4 AND PATIENT?
L6            29 S L4 NOT L5
L7             0 S L6 AND LAMINARIN?
L8             1 S L1 AND LAMINARIN?
L9             1 S LAMINARIN? (P) CYCLOPHOSPHAMIDE (P) CANCER?
L10            0 S LAMINARIN? (P) CYCLOPHOSPHAMIDE (P) TUMOR?
L11            2 S LAMINARIN? (P) CYCLOPHOSPHAMIDE
L12           18 S LAMINARIN (P) TUMOR?
L13            2 S LAMINARIN (P) TUMOR? (P) CANCER?
L14            5 S LAMINARIN (P) CANCER?
L15            3 S LAMINARIN (P) TUMOUR?
L16            1 S LAMINARIN (P) ANTINEOPLASTIC?
L17            1 S LAMINARIN (P) ANTINEOPLAS?
L18            1 S LAMINARIN (P) CHEMOTHERAP?
```

```
=> d his

     (FILE 'HOME' ENTERED AT 13:02:09 ON 07 FEB 2006)

     FILE 'CAPLUS, MEDLINE' ENTERED AT 13:02:21 ON 07 FEB 2006
L1            83 S ?GLUCAN (P) CYCLOPHOSPHAMIDE
L2            32 S ?GLUCAN (P) CYCLOPHOSPHAMIDE (P) TUMOR?
L3             9 S ?GLUCAN (P) CYCLOPHOSPHAMIDE (P) CANCER?
L4            30 S L2 NOT L3
L5             1 S L4 AND PATIENT?
L6            29 S L4 NOT L5
L7             0 S L6 AND LAMINARIN?
L8             1 S L1 AND LAMINARIN?
L9             1 S LAMINARIN? (P) CYCLOPHOSPHAMIDE (P) CANCER?
L10            0 S LAMINARIN? (P) CYCLOPHOSPHAMIDE (P) TUMOR?
L11            2 S LAMINARIN? (P) CYCLOPHOSPHAMIDE
L12           18 S LAMINARIN (P) TUMOR?
L13            2 S LAMINARIN (P) TUMOR? (P) CANCER?
L14            5 S LAMINARIN (P) CANCER?
L15            3 S LAMINARIN (P) TUMOUR?
L16            1 S LAMINARIN (P) ANTINEOPLASTIC?
L17            1 S LAMINARIN (P) ANTINEOPLAS?
L18            1 S LAMINARIN (P) CHEMOTHERAP?
```

d l20 1 ibib abs

L20 ANSWER 1 OF 1 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1987:421946 CAPLUS
DOCUMENT NUMBER: 107:21946
TITLE: Soluble phosphorylated glucan
INVENTOR(S): Diluzio, Nicholas R.
PATENT ASSIGNEE(S): Tulane Educational Fund, Inc., USA
SOURCE: PCT Int. Appl., 84 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 3
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 8701037	A1	19870226	WO 1986-US1646	19860813
W: AU, DK, FI, JP, KR, NO				
RW: AT, BE, CH, DE, FR, GB, IT, LU, NL, SE				
US 4739046	A	19880419	US 1985-767388	19850819
AU 8662296	A1	19870310	AU 1986-62296	19860813
AU 599045	B2	19900712		
EP 232405	A1	19870819	EP 1986-905497	19860813
EP 232405	B1	19920115		
R: AT, BE, CH, DE, FR, GB, IT, LI, LU, NL, SE				
JP 63500805	T2	19880324	JP 1986-504604	19860813
JP 2550332	B2	19961106		
AT 71528	E	19920215	AT 1986-905497	19860813
CA 1337408	A1	19951024	CA 1986-515890	19860813
US 4818752	A	19890404	US 1987-13298	19870210
NO 8701603	A	19870615	NO 1987-1603	19870415
NO 170586	B	19920727		
NO 170586	C	19921104		
DK 8701985	A	19870618	DK 1987-1985	19870415
FI 8701718	A	19870416	FI 1987-1718	19870416
FI 88109	B	19921231		
FI 88109	C	19930413		
US 4877777	A	19891031	US 1988-182550	19880418
PRIORITY APPLN. INFO.:			US 1985-767388 A	19850819
			EP 1986-905497 A	19860813
			WO 1986-US1646 A	19860813

AB Soluble phosphorylated glucans (I) are prepared that exhibit immunostimulation and cytostatic activities and that are useful for prophylaxis and therapy. A particulate glucan prepared from cultured Saccharomyces cerevisiae was suspended in a solution containing DMSO and urea, and reacted with H3PO4 for 6 h at 100° to yield 70-90% I. The survival rate of C3H/HeJ mice treated with immunosuppressant cortisone acetate (II) s.c. 1.5 and I i.v. 5 mg was 68% vs. 12% for the group treated with II alone. I also were effective in treating neoplastic, bacterial, viral, fungal, and parasitic diseases, and they were nontoxic, nonpyrogenic, and nonimmunogenic.

> d l28 1-4 ibib abs

L28 ANSWER 1 OF 4 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1979:66773 CAPLUS
DOCUMENT NUMBER: 90:66773
TITLE: **Antineoplastic** components of mushrooms. **Antineoplastic** activities of PS-K, a protein-bound polysaccharide of Coriolus versicolor (Fr.) Quel
AUTHOR(S): Park, Eun Kyu; Kim, Byong Kak
CORPORATE SOURCE: Coll. Pharm., Seoul Natl. Univ., Seoul, S. Korea
SOURCE: Han'guk Kyunhakhoechi (1977), 5(2), 25-30
CODEN: HKCHDD; ISSN: 0253-651X
DOCUMENT TYPE: Journal
LANGUAGE: Korean
AB **Antineoplastic** effects of PS-K, a **glucan** polysaccharide isolated from mushroom, C. versicolor, were investigated. I.p. injection of 100 mg/kg, i.m. injection of 100 mg/kg, and oral administration of 1,000 mg/kg PS-K into mice bearing sarcoma 180 showed 97.6, 78.0 and 75.9% inhibition, and PS-K also showed good results in mice bearing AH-13 and leukemia P 388. The combined use with **cyclophosphamide** [50-18-0] and vincristine [57-22-7] reduced toxic effects.

L29 ANSWER 14 OF 14 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1966:441776 CAPLUS
DOCUMENT NUMBER: 65:41776
ORIGINAL REFERENCE NO.: 65:7840d-f
TITLE: Comparative study of the biological action of polysaccharides glucan and laminarin
AUTHOR(S): Fomina, I. P.; Navashin, S. M.; Preobrazhenskaya, M. E.; Rozenfel'd, E. L.
CORPORATE SOURCE: Res. Inst. Antibiots., Moscow
SOURCE: Byulleten Eksperimental'noi Biologii i Meditsiny (1966), 61(5), 79-83
CODEN: BEBMAE; ISSN: 0365-9615
DOCUMENT TYPE: Journal
LANGUAGE: Russian

AB Albino mice were used for comparing biol. activity of glucan and **laminarin**. In the 1st series of expts., the animals were injected with glucan or **laminarin** in doses of 5-30 mg./kg., and then subjected to the action of following microorganisms: [Salmonella typhosa [Bacillus] dysenteriae sonne [Shigella sonne], Escherichia coli, Proteus mirabilis, Pseudomonas aeruginosa, Staph[ylococcus] aureus, S. albus, and D[iplococcus] pneumoniae. Glucan and **laminarin** produced nearly identical preventive effects in exptl. staphylococcic sepsis: the survival of treated animals was 76-83%, whereas the death rate of controls was 90%. In sepsis induced by gram-neg. microorganisms, glucan showed a favorable influence, while **laminarin** proved ineffective. In the 2nd exptl. series, an **antitumor** effect of both polysaccharides was observed. Glucan, 20 mg./kg. produced a pronounced **antitumor** effect against Ehrlich tumor and sarcoma 180, inhibiting their growth by 53-60%. No inhibitory activity of **laminarin** was found under analogous conditions. Since both polysaccharides were of the same mol. structure, it is suggested that different biol. activities was due to their size and configuration. This explanation is supported by a reduction in glucan biol. activity after splitting off some of its glucose residues.

L29 ANSWER 11 OF 14 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1996:385442 CAPLUS
DOCUMENT NUMBER: 125:75581
TITLE: Effect of highly branched (1 → 3)-β-D-glucan, OL-2, on zymosan-mediated hydrogen peroxide production by murine peritoneal macrophages
AUTHOR(S): Chiba, Norihisa; Ohno, Naohito; Terui, Takayoshi; Adachi, Yoshiyuki; Yadomae, Toshiro
CORPORATE SOURCE: Lab. Immunopharmacol. Microbial Products, School Pharmacy, Tokyo Univ. Pharmacy Life Sci., Tokyo, 192-03, Japan
SOURCE: Pharmaceutical and Pharmacological Letters (1996), 6(1), 12-15
CODEN: PPLEE3; ISSN: 0939-9488
PUBLISHER: Medpharm Scientific Publishers
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Degree of branching is an important contributing factor to define immunopharmacol. activity of (1→6)-branched (1→3)-β-D-glucans. OL-2 is a highly branched (1→3)-β-D-glucan showing low **antitumor** activity and high hematopoietic activity. In this paper, we examined effect of OL-2 on zymosan, a particulate β-glucan, mediated H2O2 production by murine peritoneal macrophages (PEM) and compared the activity with other glucans. We used the scopoletin fluorescence assay to measure production of H2O2. The glucans used were **laminarin** (linear), SPG (branched, degree of branching is 1/3), GRN (branched, 1/3), SSG (branched, 1/2), and OL-2 (branched, 2/3). Pretreatment of proteose peptone elicited PEM with OL-2 for 6 h at 37° inhibited the subsequent zymosan-mediated H2O2 production similar to others. Macrophages elicited by i.p. administration of soluble β-glucans increased zymosan-mediated H2O2 production compared with control group, but the strength of the effect was different among glucans (OL-2 > SSG > GRN). Similar results were observed all the strains of ICR, BALB/c, C3H/HeN, AKR. **Antitumor** activity of β-glucan was high in the former two strains. These facts strongly suggested that the structure-activity relation of the glucan induced H2O2 production was not strongly correlated with that of **antitumor** activity.

```
=> d his

     (FILE 'HOME' ENTERED AT 13:02:09 ON 07 FEB 2006)

     FILE 'CAPLUS, MEDLINE' ENTERED AT 13:02:21 ON 07 FEB 2006
L1            83 S ?GLUCAN (P) CYCLOPHOSPHAMIDE
L2            32 S ?GLUCAN (P) CYCLOPHOSPHAMIDE (P) TUMOR?
L3             9 S ?GLUCAN (P) CYCLOPHOSPHAMIDE (P) CANCER?
L4            30 S L2 NOT L3
L5             1 S L4 AND PATIENT?
L6            29 S L4 NOT L5
L7             0 S L6 AND LAMINARIN?
L8             1 S L1 AND LAMINARIN?
L9             1 S LAMINARIN? (P) CYCLOPHOSPHAMIDE (P) CANCER?
L10            0 S LAMINARIN? (P) CYCLOPHOSPHAMIDE (P) TUMOR?
L11            2 S LAMINARIN? (P) CYCLOPHOSPHAMIDE
L12           18 S LAMINARIN (P) TUMOR?
L13            2 S LAMINARIN (P) TUMOR? (P) CANCER?
L14            5 S LAMINARIN (P) CANCER?
L15            3 S LAMINARIN (P) TUMOUR?
L16            1 S LAMINARIN (P) ANTINEOPLASTIC?
L17            1 S LAMINARIN (P) ANTINEOPLAS?
L18            1 S LAMINARIN (P) CHEMOTHERAP?
L19           11 S ?GLUCANS (P) CYCLOPHOSPHAMIDE
L20            1 S L19 NOT L1
L21           51 S L1 NOT L2
L22           44 S L21 NOT L3
L23            1 S L22 AND COMPOSITION?
L24            2 S L22 AND PATIENT?
L25            3 S L22 AND CHEMO?
L26           41 S L22 NOT L25
L27           39 S L26 NOT L24
L28            4 S L27 AND ANTINEOP?
L29           14 S LAMINARIN? (P) ANTITUMOR?
```

```
=> d his

     (FILE 'HOME' ENTERED AT 13:02:09 ON 07 FEB 2006)

     FILE 'CAPLUS, MEDLINE' ENTERED AT 13:02:21 ON 07 FEB 2006
L1            83 S ?GLUCAN (P) CYCLOPHOSPHAMIDE
L2            32 S ?GLUCAN (P) CYCLOPHOSPHAMIDE (P) TUMOR?
L3             9 S ?GLUCAN (P) CYCLOPHOSPHAMIDE (P) CANCER?
L4            30 S L2 NOT L3
L5             1 S L4 AND PATIENT?
L6            29 S L4 NOT L5
L7             0 S L6 AND LAMINARIN?
L8             1 S L1 AND LAMINARIN?
L9             1 S LAMINARIN? (P) CYCLOPHOSPHAMIDE (P) CANCER?
L10            0 S LAMINARIN? (P) CYCLOPHOSPHAMIDE (P) TUMOR?
L11            2 S LAMINARIN? (P) CYCLOPHOSPHAMIDE
L12           18 S LAMINARIN (P) TUMOR?
L13            2 S LAMINARIN (P) TUMOR? (P) CANCER?
L14            5 S LAMINARIN (P) CANCER?
L15            3 S LAMINARIN (P) TUMOUR?
L16            1 S LAMINARIN (P) ANTINEOPLASTIC?
L17            1 S LAMINARIN (P) ANTINEOPLAS?
L18            1 S LAMINARIN (P) CHEMOTHERAP?
L19           11 S ?GLUCANS (P) CYCLOPHOSPHAMIDE
L20            1 S L19 NOT L1
L21           51 S L1 NOT L2
L22           44 S L21 NOT L3
L23            1 S L22 AND COMPOSITION?
L24            2 S L22 AND PATIENT?
L25            3 S L22 AND CHEMO?
L26           41 S L22 NOT L25
L27           39 S L26 NOT L24
L28            4 S L27 AND ANTINEOP?
L29           14 S LAMINARIN? (P) ANTITUMOR?
```

```
=> s 9012-72-0
L1            1 9012-72-0
                (9012-72-0/RN)

=> d l1

L1   ANSWER 1 OF 1  REGISTRY  COPYRIGHT 2006 ACS on STN
RN   9012-72-0  REGISTRY
ED   Entered STN:  16 Nov 1984
CN   D-Glucan (9CI)  (CA INDEX NAME)
OTHER NAMES:
CN   D-Glucosan
CN   Glucan
CN   Glucosan
CN   Poly-D-glucan
CN   Polyglucan
CN   Polyglucosan
DR   9037-91-6, 9072-21-3
MF   Unspecified
CI   PMS, COM, MAN
PCT  Manual registration
LC   STN Files:   ADISNEWS, AGRICOLA, ANABSTR, BIOSIS, BIOTECHNO, CA, CAPLUS,
       CHEMCATS, CHEMLIST, CIN, CSCHEM, DDFU, DIOGENES, DRUGU, EMBASE, IFICDB,
       IFIPAT, IFIUDB, IPA, MEDLINE, NAPRALERT, NIOSHTIC, PIRA, PROMT, RTECS*,
       TOXCENTER, USPAT2, USPATFULL
         (*File contains numerically searchable property data)
     Other Sources:   EINECS**, NDSL**
         (**Enter CHEMLIST File for up-to-date regulatory information)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***

**PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT**

            2443 REFERENCES IN FILE CA (1907 TO DATE)
             183 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
            2446 REFERENCES IN FILE CAPLUS (1907 TO DATE)
```

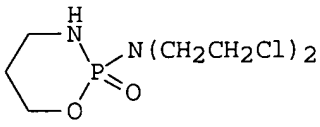
L12 ANSWER 15 OF 18 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1966:441776 CAPLUS
DOCUMENT NUMBER: 65:41776
ORIGINAL REFERENCE NO.: 65:7840d-f
TITLE: Comparative study of the biological action of polysaccharides glucan and laminarin
AUTHOR(S): Fomina, I. P.; Navashin, S. M.; Preobrazhenskaya, M. E.; Rozenfel'd, E. L.
CORPORATE SOURCE: Res. Inst. Antibiots., Moscow
SOURCE: Byulleten Eksperimental'noi Biologii i Meditsiny (1966), 61(5), 79-83
CODEN: BEBMAE; ISSN: 0365-9615
DOCUMENT TYPE: Journal
LANGUAGE: Russian

AB Albino mice were used for comparing biol. activity of glucan and **laminarin**. In the 1st series of expts., the animals were injected with glucan or **laminarin** in doses of 5-30 mg./kg., and then subjected to the action of following microorganisms: [Salmonella typhosa [Bacillus] dysenteriae sonne [Shigella sonne], Escherichia coli, Proteus mirabilis, Pseudomonas aeruginosa, Staph[ylococcus] aureus, S. albus, and D[iplococcus] pneumoniae. Glucan and **laminarin** produced nearly identical preventive effects in exptl. staphylococcic sepsis: the survival of treated animals was 76-83%, whereas the death rate of controls was 90%. In sepsis induced by gram-neg. microorganisms, glucan showed a favorable influence, while **laminarin** proved ineffective. In the 2nd exptl. series, an antitumor effect of both polysaccharides was observed. Glucan, 20 mg./kg. produced a pronounced antitumor effect against Ehrlich **tumor** and sarcoma 180, inhibiting their growth by 53-60%. No inhibitory activity of **laminarin** was found under analogous conditions. Since both polysaccharides were of the same mol. structure, it is suggested that different biol. activities was due to their size and configuration. This explanation is supported by a reduction in glucan biol. activity after splitting off some of its glucose residues.

L12 ANSWER 15 OF 18 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1966:441776 CAPLUS
DOCUMENT NUMBER: 65:41776
ORIGINAL REFERENCE NO.: 65:7840d-f
TITLE: Comparative study of the biological action of polysaccharides glucan and laminarin
AUTHOR(S): Fomina, I. P.; Navashin, S. M.; Preobrazhenskaya, M. E.; Rozenfel'd, E. L.
CORPORATE SOURCE: Res. Inst. Antibiots., Moscow
SOURCE: Byulleten Eksperimental'noi Biologii i Meditsiny (1966), 61(5), 79-83
CODEN: BEBMAE; ISSN: 0365-9615
DOCUMENT TYPE: Journal
LANGUAGE: Russian

AB Albino mice were used for comparing biol. activity of glucan and **laminarin**. In the 1st series of expts., the animals were injected with glucan or **laminarin** in doses of 5-30 mg./kg., and then subjected to the action of following microorganisms: [Salmonella typhosa [Bacillus] dysenteriae sonne [Shigella sonne], Escherichia coli, Proteus mirabilis, Pseudomonas aeruginosa, Staph[ylococcus] aureus, S. albus, and D[iplococcus] pneumoniae. Glucan and **laminarin** produced nearly identical preventive effects in exptl. staphylococcic sepsis: the survival of treated animals was 76-83%, whereas the death rate of controls was 90%. In sepsis induced by gram-neg. microorganisms, glucan showed a favorable influence, while **laminarin** proved ineffective. In the 2nd exptl. series, an antitumor effect of both polysaccharides was observed. Glucan, 20 mg./kg. produced a pronounced antitumor effect against Ehrlich **tumor** and sarcoma 180, inhibiting their growth by 53-60%. No inhibitory activity of **laminarin** was found under analogous conditions. Since both polysaccharides were of the same mol. structure, it is suggested that different biol. activities was due to their size and configuration. This explanation is supported by a reduction in glucan biol. activity after splitting off some of its glucose residues.

L3 ANSWER 7 OF 9 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1978:453298 CAPLUS
DOCUMENT NUMBER: 89:53298
TITLE: The synergistic effect of cyclophosphamide and glucan on experimental acute myelogenous and lymphocytic leukemia
AUTHOR(S): Di Luzio, N. R.; Cook, J. A.; Cohen, C.; Rodrigue, J.; Jones, E.
CORPORATE SOURCE: Dep. Physiol., Tulane Univ. Sch. Med., New Orleans, LA, USA
SOURCE: Proc. EURES Symp. Macrophage Cancer (1977), 188-201. Editor(s): James, Keith; McBride, Bill; Stuart, Angus. Univ. Edinburgh, Med. Sch.: Edinburgh, Scot. CODEN: 38BZA9
DOCUMENT TYPE: Conference
LANGUAGE: English
GI



I

AB In rats with Shay myelogenous leukemia, primary tumor growth was significantly reduced after administration of either **cyclophosphamide** (I) [50-18-0] (40 mg/kg, i.p., on days 3 and 6) or **glucan** [9012-72-0] (10 mg/kg, i.v. on days 3 and 6) alone compared to control rats. The most effective antineoplastic action, however, was evident with concurrent **glucan** and I therapy as denoted by a mean 97% decrease in tumor weight compared to control rats. In mice, increased survival after i.v. administered acute myelogenous leukemic cells was also observed in the **glucan** and I-treated group. I inhibited, to some degree, the **glucan**-induced hepatic granuloma. The degree of hepatic metastases was significantly reduced in both rats and mice by the conjoint use of I and **glucan**. Thus, **glucan** may be a valuable adjunct to conventional **cancer** chemotherapy.

L3 ANSWER 5 OF 9 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1998:803767 CAPLUS
DOCUMENT NUMBER: 130:204804
TITLE: In vitro and in vivo hematopoietic activities of Betafectin PGG-glucan
AUTHOR(S): Patchen, Myra L.; Vaudrain, Tracy; Correira, Heidi; Martin, Tracey; Reese, Debrah
CORPORATE SOURCE: Alpha-Beta Technology, Worcester, MA, USA
SOURCE: Experimental Hematology (Charlottesville, Virginia) (1998), 26(13), 1247-1254
CODEN: EXHMA6; ISSN: 0301-472X
PUBLISHER: Carden Jennings Publishing
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Betafectin PGG-glucan is a novel β-(1,3)glucan that has broad-spectrum anti-infective activities without cytokine induction. Here the authors report that PGG-glucan also has both in vitro and in vivo hematopoietic activities. In vitro studies with bone marrow target cells from the C3H/HeN mouse revealed that although PGG-glucan alone had no direct effect on hematopoietic colony-forming cell (CFC) growth, when combined with granulocyte colony-stimulating factor (CSF) or granulocyte-macrophage CSF, it increased CFC nos. 1.5- to 2.0-fold over those obtained with CSFs alone. Bone marrow cells cultured for high-proliferative-potential CFCs in the presence of interleukin (IL)-1, IL-3, macrophage CSF, and stem cell factor (SCF), or cultured for erythroid burst-forming units in the presence of IL-3, SCF, and erythropoietin, also exhibited enhanced growth in the presence of PGG-glucan. The synergistic effect of PGG-glucan was specific and could be abrogated by anti-PGG-glucan antibody. The ability of PGG-glucan to modulate hematopoiesis in vivo was evaluated in myelosuppressed rodents and primates. C3H/HeN female mice were i.v. administered saline solution or PGG-glucan (0.5 mg/kg) 24 h before the i.p. administration of cyclophosphamide (200 mg/kg), and the recovery of bone marrow cellularity and granulocyte-macrophage progenitor cells was evaluated on days 4 and 8 after cyclophosphamide treatment. At both time points, enhanced hematopoietic recovery was observed in PGG-glucan-treated mice compared with saline-treated control mice. In a final series of in vivo expts., the authors evaluated the ability of therapeutically administered PGG-glucan to enhance hematopoietic recovery in cyclophosphamide-treated cynomolgus monkeys. Monkeys received i.v. infusions of cyclophosphamide (55 mg/kg) on days 1 and 2, followed on days 3 and 10 by i.v. infusion of PGG-glucan (0.5, 1.0, or 2.0 mg/kg). Compared with those in saline-treated monkeys, accelerated white blood cell recovery and a reduction in the median duration of neutropenia were observed in PGG-glucan-treated monkeys. These studies illustrate that PGG-glucan has both in vitro and in vivo hematopoietic activities and that this agent may be useful in the prevention and/or treatment of chemotherapy-associated myelosuppression.

REFERENCE COUNT: 45 THERE ARE 45 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 4 OF 9 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2000:324986 CAPLUS
DOCUMENT NUMBER: 133:202741
TITLE: Induction of apoptosis in human prostatic cancer cells with β-glucan (Maitake mushroom polysaccharide)
AUTHOR(S): Fullerton, Sean A.; Samadi, Albert A.; Tortorelis, Dean G.; Choudhury, Muhammad S.; Mallouh, Camille; Tazaki, Hiroshi; Konno, Sensuke
CORPORATE SOURCE: Department of Urology, New York Medical College, Valhalla, NY, USA
SOURCE: Molecular Urology (2000), 4(1), 7-13
CODEN: MOURFE; ISSN: 1091-5362
PUBLISHER: Mary Ann Liebert, Inc.
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Human prostate cancer PC-3 cells were treated with various concns. of the highly purified β-glucan preparation Grifron-D (GD), and viability was determined after 24 h. Lipid peroxidn. (LPO) assay and in situ hybridization (ISH) were performed to evaluate the antitumor mechanism of GD. A concentration-response study showed that almost complete (>95%) cell death was attained in 24 h with GD ≥480 μg/mL. Combinations of GD in a concentration as low as 30-60 μg/mL with 200 μM vitamin C were as effective as GD alone at 480 μg/mL, inducing >90% cytotoxic cell death. Simultaneous use with various anticancer drugs showed little potentiation of their efficacy, except for the carmustine/GD combination (.apprx.90% reduction in cell viability). The 2-fold elevated LPO level and pos. ISH staining of GD-treated cells indicated oxidative membrane damage resulting in apoptotic cell death. Thus, a bioactive β-glucan from the Maitake mushroom has a cytotoxic effect, presumably through oxidative stress, on prostatic cancer cells in vitro, leading to apoptosis. Potentiation of GD action by vitamin C and the chemosensitizing effect of GD on carmustine may also have clin. implications. This unique mushroom polysaccharide may have potential as an alternative therapeutic modality for prostate cancer.
REFERENCE COUNT: 26 THERE ARE 26 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 3 OF 9 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2003:808828 CAPLUS
DOCUMENT NUMBER: 140:138897
TITLE: β-Glucan inhibits the genotoxicity of cyclophosphamide, adriamycin and cisplatin
AUTHOR(S): Tohamy, Amany A.; El-Ghor, Akmal A.; El-Nahas, Soheir M.; Noshy, Magda M.
CORPORATE SOURCE: Faculty of Science, Zoology Department, Helwan University, Cairo, Egypt
SOURCE: Mutation Research (2003), 541(1-2), 45-53
CODEN: MUREAV; ISSN: 0027-5107
PUBLISHER: Elsevier Science B.V.
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The inhibitory effects of β- **glucan** (βG), one of the biol. response modifiers, on the induction of chromosomal aberrations in the bone marrow and spermatogonial cells of mice treated with various anti-neoplastic drugs were investigated. β- **Glucan** (100 mg/kg bw, i.p.) pre-treatment reduced the total number of cells with structural chromosomal aberrations scored after the treatment with **cyclophosphamide** (CP) (2.5 mg/kg bw, i.p.) adriamycin (ADR) (12 mg/kg bw, i.p.) and cis-diamminedichloroplatinum-II (cisplatin) (5 mg/kg bw, i.p.) by about 41.1, 26.9 and 57.7% in bone marrow and 44.4, 55 and 57.1% in spermatogonial cells, resp. This protective effect of β- **glucan** could be attributed to its scavenging ability to trap free-radicals produced during the biotransformation of these anti-neoplastic drugs. β- **Glucan** also markedly restored the mitotic activity of bone marrow cells that had been suppressed by the anti-neoplastic drugs. These results indicate that in addition to the known immunopotentiating activity of β- **glucan**, it plays a role in reducing genotoxicity induced by anti-neoplastic drugs during **cancer** chemotherapy.

REFERENCE COUNT: 48 THERE ARE 48 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 1 OF 1 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1992:503739 CAPLUS
DOCUMENT NUMBER: 117:103739
TITLE: Suppressing effects of glucan on micronuclei induced by cyclophosphamide in mice
AUTHOR(S): Chorvatovicova, Darina; Navarova, Jana
CORPORATE SOURCE: Inst. Ecobiol., Slovak Acad. Sci., Bratislava, 814 34, Czech.
SOURCE: Mutation Research (1992), 282(3), 147-50
CODEN: MUREAV; ISSN: 0027-5107
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The effect of pretreatment with **carboxymethylglucan** (CMG) on the frequency of micronuclei induced by **cyclophosphamide** administration in mice was evaluated. Two doses of CMG (50 mg/kg) injected either i.p. 24 h or i.v. 1 h prior to two **cyclophosphamide** administrations (80 mg/kg) significantly decreased the frequency of micronucleated PCE in bone marrow. Of two evaluated derivs. of **carboxymethylglucan**, the K3 derivative was most efficient. The results show that it is possible to achieve a suppressive effect of soluble **carboxymethylglucan** prepared from Saccharomyces cerevisiae against **cyclophosphamide** mutagenicity. The notion may be useful for **glucan**'s effects against pharmacocarcinogenesis. Therapeutic application of **glucan** with **cyclophosphamide** therapy may provide a remarkable decrease of the secondary **tumor** risk. The utilization of these results for human **patients** needs to be considered.

L12 ANSWER 17 OF 18 MEDLINE on STN
ACCESSION NUMBER: 1999426885 MEDLINE
DOCUMENT NUMBER: PubMed ID: 10495437
TITLE: Inhibition of heparanase activity and **tumor** metastasis by **laminarin** sulfate and synthetic phosphorothioate oligodeoxynucleotides.
AUTHOR: Miao H Q; Elkin M; Aingorn E; Ishai-Michaeli R; Stein C A; Vlodavsky I
CORPORATE SOURCE: Department of Oncology, Hadassah University Hospital, Jerusalem, Israel.
SOURCE: International journal of cancer. Journal international du cancer, (1999 Oct 29) 83 (3) 424-31.
Journal code: 0042124. ISSN: 0020-7136.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199910
ENTRY DATE: Entered STN: 19991101
Last Updated on STN: 19991101
Entered Medline: 19991021

AB Heparanase activity correlates with the metastatic potential of **tumor** cells. Moreover, the anti-metastatic effect of non-anti-coagulant species of heparin and certain sulfated polysaccharides was attributed to their heparanase-inhibiting activity. We investigated the effect of a chemically sulfated polysaccharide (**laminarin**), consisting primarily of beta-1,3 glucan (sodium **laminarin**), and of synthetic phosphorothioate oligodeoxynucleotides, primarily phosphorothioate homopolymer of cytidine (SdC28), on heparanase activity and **tumor** metastasis. Investigation of the ability of **tumor** cells to degrade heparan sulfate in intact extracellular matrix revealed that heparanase activity expressed by B16-BL6 mouse melanoma cells and 13762 MAT rat mammary adenocarcinoma cells was effectively inhibited by LS (50% inhibition at 0.2-1 microgram/ml), but there was no inhibition by sodium **laminarin** up to a concentration of 50 microgram/ml. Complete inhibition of the melanoma heparanase was obtained in the presence of 0.1 microM SdC28. A single i.p. injection of **laminarin** sulfate, but not of sodium **laminarin**, before i.v. inoculation of the melanoma or breast-carcinoma cells inhibited the extent of lung colonization by the **tumor** cells by 80 to 90%. Similar inhibition was exerted by 0.1 microM SdC28. At the effective concentrations, both compounds had a small effect on proliferation of the **tumor** cells and on growth of the primary **tumors** in vivo. These results further emphasize the involvement of heparanase in **tumor** metastasis and the potential clinical application of diverse heparanase-inhibiting molecules such as sulfated polysaccharides and synthetic polyanionic molecules.

L12 ANSWER 16 OF 18 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1963:444312 CAPLUS
DOCUMENT NUMBER: 59:44312
ORIGINAL REFERENCE NO.: 59:8030h
TITLE: Effects of sulfated degraded **laminarin** on experimental **tumor** growth
AUTHOR(S): Jolles, B.; Remington, Mary; Andrews, P. S.
CORPORATE SOURCE: Gen. Hosp., Northampton, UK
SOURCE: British Journal of Cancer (1963), 17, 109-15
CODEN: BJCAAI; ISSN: 0007-0920
DOCUMENT TYPE: Journal
LANGUAGE: Unavailable
AB The compound, a polysaccharide derivative, inhibited the growth of sarcoma 180 when injected at the site of the transplant or into growing tumors.

L12 ANSWER 11 OF 18 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1996:423114 CAPLUS
DOCUMENT NUMBER: 125:131856
TITLE: Inhibition of angiogenesis and murine **tumor** growth by **laminarin** sulfate
AUTHOR(S): Hoffman, R.; Paper, D. H.; Donaldson, J.; Vogl, H.
CORPORATE SOURCE: Clinical Oncology and Radiotherapeutics Unit, MRC Centre, Cambridge, CB2 2QH, UK
SOURCE: British Journal of Cancer (1996), 73(10), 1183-1186
CODEN: BJCAAI; ISSN: 0007-0920
PUBLISHER: Stockton
DOCUMENT TYPE: Journal
LANGUAGE: English
AB LAM S5 is a polysulfated derivative of the glucan **laminarin** that inhibits basic fibroblast growth factor (bFGF) binding and the bFGF-stimulated proliferation of fetal bovine heart endothelial (FBHE) cells. This report demonstrates that LAM S5 has anti-angiogenic activity, as shown by inhibition of tubule formation by endothelial cells cultured on Matrigel and inhibition of vascularization of the chick chorioallantoic membrane. In addition, LAM S5 caused a **tumor** growth delay of the murine RIF-1 **tumor** of 2.6 days.

d 120 1 ibib abs

L20 ANSWER 1 OF 1 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1987:421946 CAPLUS
DOCUMENT NUMBER: 107:21946
TITLE: Soluble phosphorylated glucan
INVENTOR(S): Diluzio, Nicholas R.
PATENT ASSIGNEE(S): Tulane Educational Fund, Inc., USA
SOURCE: PCT Int. Appl., 84 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 3
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 8701037	A1	19870226	WO 1986-US1646	19860813
W: AU, DK, FI, JP, KR, NO				
RW: AT, BE, CH, DE, FR, GB, IT, LU, NL, SE				
US 4739046	A	19880419	US 1985-767388	19850819
AU 8662296	A1	19870310	AU 1986-62296	19860813
AU 599045	B2	19900712		
EP 232405	A1	19870819	EP 1986-905497	19860813
EP 232405	B1	19920115		
R: AT, BE, CH, DE, FR, GB, IT, LI, LU, NL, SE				
JP 63500805	T2	19880324	JP 1986-504604	19860813
JP 2550332	B2	19961106		
AT 71528	E	19920215	AT 1986-905497	19860813
CA 1337408	A1	19951024	CA 1986-515890	19860813
US 4818752	A	19890404	US 1987-13298	19870210
NO 8701603	A	19870615	NO 1987-1603	19870415
NO 170586	B	19920727		
NO 170586	C	19921104		
DK 8701985	A	19870618	DK 1987-1985	19870415
FI 8701718	A	19870416	FI 1987-1718	19870416
FI 88109	B	19921231		
FI 88109	C	19930413		
US 4877777	A	19891031	US 1988-182550	19880418

PRIORITY APPLN. INFO.:	US 1985-767388	A	19850819
	EP 1986-905497	A	19860813
	WO 1986-US1646	A	19860813

AB Soluble phosphorylated glucans (I) are prepared that exhibit immunostimulation and cytostatic activities and that are useful for prophylaxis and therapy. A particulate glucan prepared from cultured Saccharomyces cerevisiae was suspended in a solution containing DMSO and urea, and reacted with H3PO4 for 6 h at 100° to yield 70-90% I. The survival rate of C3H/HeJ mice treated with immunosuppressant cortisone acetate (II) s.c. 1.5 and I i.v. 5 mg was 68% vs. 12% for the group treated with II alone. I also were effective in treating neoplastic, bacterial, viral, fungal, and parasitic diseases, and they were nontoxic, nonpyrogenic, and nonimmunogenic.

> d l28 1-4 ibib abs

L28 ANSWER 1 OF 4 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1979:66773 CAPLUS
DOCUMENT NUMBER: 90:66773
TITLE: **Antineoplastic** components of mushrooms. **Antineoplastic** activities of PS-K, a protein-bound polysaccharide of Coriolus versicolor (Fr.) Quel
AUTHOR(S): Park, Eun Kyu; Kim, Byong Kak
CORPORATE SOURCE: Coll. Pharm., Seoul Natl. Univ., Seoul, S. Korea
SOURCE: Han'guk Kyunhakhoechi (1977), 5(2), 25-30
CODEN: HKCHDD; ISSN: 0253-651X
DOCUMENT TYPE: Journal
LANGUAGE: Korean
AB **Antineoplastic** effects of PS-K, a **glucan** polysaccharide isolated from mushroom, C. versicolor, were investigated. I.p. injection of 100 mg/kg, i.m. injection of 100 mg/kg, and oral administration of 1,000 mg/kg PS-K into mice bearing sarcoma 180 showed 97.6, 78.0 and 75.9% inhibition, and PS-K also showed good results in mice bearing AH-13 and leukemia P 388. The combined use with **cyclophosphamide** [50-18-0] and vincristine [57-22-7] reduced toxic effects.

L29 ANSWER 14 OF 14 CAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 1966:441776 CAPLUS
DOCUMENT NUMBER: 65:41776
ORIGINAL REFERENCE NO.: 65:7840d-f
TITLE: Comparative study of the biological action of polysaccharides glucan and laminarin
AUTHOR(S): Fomina, I. P.; Navashin, S. M.; Preobrazhenskaya, M. E.; Rozenfel'd, E. L.
CORPORATE SOURCE: Res. Inst. Antibiots., Moscow
SOURCE: Byulleten Eksperimental'noi Biologii i Meditsiny (1966), 61(5), 79-83
CODEN: BEBMAE; ISSN: 0365-9615
DOCUMENT TYPE: Journal
LANGUAGE: Russian

AB Albino mice were used for comparing biol. activity of glucan and **laminarin**. In the 1st series of expts., the animals were injected with glucan or **laminarin** in doses of 5-30 mg./kg., and then subjected to the action of following microorganisms: [Salmonella typhosa [Bacillus] dysenteriae sonne [Shigella sonne], Escherichia coli, Proteus mirabilis, Pseudomonas aeruginosa, Staph[ylococcus] aureus, S. albus, and D[iplococcus] pneumoniae. Glucan and **laminarin** produced nearly identical preventive effects in exptl. staphylococcic sepsis: the survival of treated animals was 76-83%, whereas the death rate of controls was 90%. In sepsis induced by gram-neg. microorganisms, glucan showed a favorable influence, while **laminarin** proved ineffective. In the 2nd exptl. series, an **antitumor** effect of both polysaccharides was observed. Glucan, 20 mg./kg. produced a pronounced **antitumor** effect against Ehrlich tumor and sarcoma 180, inhibiting their growth by 53-60%. No inhibitory activity of **laminarin** was found under analogous conditions. Since both polysaccharides were of the same mol. structure, it is suggested that different biol. activities was due to their size and configuration. This explanation is supported by a reduction in glucan biol. activity after splitting off some of its glucose residues.

L29 ANSWER 11 OF 14 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1996:385442 CAPLUS
DOCUMENT NUMBER: 125:75581
TITLE: Effect of highly branched (1 → 3)-β-D-glucan, OL-2, on zymosan-mediated hydrogen peroxide production by murine peritoneal macrophages
AUTHOR(S): Chiba, Norihisa; Ohno, Naohito; Terui, Takayoshi; Adachi, Yoshiyuki; Yadomae, Toshiro
CORPORATE SOURCE: Lab. Immunopharmacol. Microbial Products, School Pharmacy, Tokyo Univ. Pharmacy Life Sci., Tokyo, 192-03, Japan
SOURCE: Pharmaceutical and Pharmacological Letters (1996), 6(1), 12-15
CODEN: PPLEE3; ISSN: 0939-9488
PUBLISHER: Medpharm Scientific Publishers
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Degree of branching is an important contributing factor to define immunopharmacol. activity of (1→6)-branched (1→3)-β-D-glucans. OL-2 is a highly branched (1→3)-β-D-glucan showing low **antitumor** activity and high hematopoietic activity. In this paper, we examined effect of OL-2 on zymosan, a particulate β-glucan, mediated H2O2 production by murine peritoneal macrophages (PEM) and compared the activity with other glucans. We used the scopoletin fluorescence assay to measure production of H2O2. The glucans used were **laminarin** (linear), SPG (branched, degree of branching is 1/3), GRN (branched, 1/3), SSG (branched, 1/2), and OL-2 (branched, 2/3). Pretreatment of proteose peptone elicited PEM with OL-2 for 6 h at 37° inhibited the subsequent zymosan-mediated H2O2 production similar to others. Macrophages elicited by i.p. administration of soluble β-glucans increased zymosan-mediated H2O2 production compared with control group, but the strength of the effect was different among glucans (OL-2 > SSG > GRN). Similar results were observed all the strains of ICR, BALB/c, C3H/HeN, AKR. **Antitumor** activity of β-glucan was high in the former two strains. These facts strongly suggested that the structure-activity relation of the glucan induced H2O2 production was not strongly correlated with that of **antitumor** activity.

L5 ANSWER 3 OF 18 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2003:434382 CAPLUS
DOCUMENT NUMBER: 139:12302
TITLE: Laminaria polysaccharides for therapeutical treatments
INVENTOR(S): Yvin, Jean-Claude; Vetvicka, Vaclav
PATENT ASSIGNEE(S): Laboratoires Goeemar S.A., Fr.
SOURCE: PCT Int. Appl., 29 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2003045414	A2	20030605	WO 2002-EP13512	20021129
WO 2003045414	A3	20031016		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SC, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZW				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
US 2003119780	A1	20030626	US 2001-999202	20011130
US 6660722	B2	20031209		
CA 2468314	AA	20030605	CA 2002-2468314	20021129
AU 2002352187	A1	20030610	AU 2002-352187	20021129
EP 1448215	A2	20040825	EP 2002-787872	20021129
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, SK				
JP 2005510543	T2	20050421	JP 2003-546915	20021129

PRIORITY APPLN. INFO.:
US 2001-999202 A 20011130
WO 2002-EP13512 W 20021129

AB A therapeutical method comprises administration to a patient of an effective amount of especially soluble **laminarin** for the treatment of **tumors** and more generally of cancers of the group comprising breast cancer, lung cancer, esophagus cancer, stomach cancer, intestine and colon cancers, and for the treatment of viral, bacterial and fungal diseases as well as diseases related to immunostimulant deficiencies of human beings and warm-blood animals.

L5 ANSWER 8 OF 18 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1999:773732 CAPLUS
DOCUMENT NUMBER: 132:288446
TITLE: Activation of murine peritoneal macrophages by laminarin
AUTHOR(S): Xue, Jingbo; Liu, Xiying; Zhang, Hongfen
CORPORATE SOURCE: Medical College, Qingdao University, Tsingtao, 266021, Peop. Rep. China
SOURCE: Zhongguo Haiyang Yaowu (1999), 18(3), 23-25
CODEN: ZHYAE8; ISSN: 1002-3461
PUBLISHER: Shandongsheng Haiyang Yaowu Kexue Yanjiuso
DOCUMENT TYPE: Journal
LANGUAGE: Chinese

AB Activation of murine peritoneal macrophages by laminarin was studied in G57BL/6 mice. Peritoneal macrophages could be markedly activated by i.p. injection of laminarin (40 mg/kg) for cytolysis. Laminarin activated peritoneal macrophages secretion of TNF in vitro in the presence of LPS (10 ng/mL).